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RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
37TH YEAR—ESTABLISHED 1956

- Editorial: What Are You Doing With All Those Numbers?
- Evaluation of MDI Spacers and Adapters: Their Effect on the Respirable Volume of Medication
- Evaluation of the Ability of the Syncoxy Breath-Synchronized Valve To Provide Adequate Oxygen Levels
- An Alternative Method for Delivery of Ribavirin to Nonventilated Pediatric Patients
- More Clinical Practice Guidelines: Now What?
- *Letters*: ACLS, F_{DO_2}, Spiders, BiPAP, and Sighs

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effectively in child
However, because of t
inability of many pediatric patients
younger than 10 years of age to
coordinate the actuation of the MDI, a
holding chamber should always be used. A
holding chamber with mask should be used in those
less than 3 years of age."
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" .... a holding chamber should be used with inhaled steroids
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American Physicians Consider Euthanasia (editorial)—A Hastillo, SM Ayres. Crit Care Med 1992;20(5):566. (Pertains to Caralis et al paper abstracted on Page 843.)

Positioning and SIDS (recommendation)—American Academy of Pediatrics Task Force on Infant Positioning and SIDS. Pediatrics 1992;89:1120.

Recent Developments in Pulse Oximetry (review)—JW Severinghaus, JF Kelleher. Anesthesiology 1992;76:1018-1038.

Adenosine during Cardiac Arrest and Cardiopulmonary Resuscitation: A Placebo-Controlled, Randomized Trial—M von Planta, I von Planta, O Wagner, D Scheidegger. Crit Care Med 1992;20:645.

BACKGROUND & HYPOTHESIS TESTED: The effects of adenosine (100 μg/kg/min; n = 7) were examined during rodent cardiopulmonary resuscitation (CPR). Changes in coronary artery perfusion pressure, end-tidal P_{CO_2}, and arterial acid-base status of anesthetized, male, Sprague-Dawley rats were compared with CPR controls (0.9% sodium chloride; n = 7) and with sham controls (n = 9). Sustained ventricular fibrillation was induced and precordial chest compression was followed by defibrillation. RESULTS: After 6 mins of cardiac arrest, 6 (86%) of 7 adenosine-treated animals were resuscitated after adenosine infusion and 4 (57%) of 7 control animals were resuscitated after sodium chloride infusion. During chest compression, coronary artery perfusion pressure was 7 ± 2 torr after adenosine, but was 22 ± 3 torr in the controls ($p < 0.01$). Parallel decreases were observed in mean aortic pressure. Arterial and end-tidal P_{CO_2} significantly ($p < 0.01$) decreased after adenosine. These changes contrasted with a second control group of 9 identically prepared animals which, in the absence of ventricular fibrillation and subsequent chest compression, demonstrated no changes in hemodynamic, respiratory, or blood gas variables. CONCLUSIONS: Adenosine decreased coronary artery perfusion pressure. However, despite marked reductions in coronary artery perfusion pressure, survival was not compromised after adenosine infusion in this rodent model of CPR.

Increased Sensitivity to Mechanical Ventilation after Surfactant Inactivation in Young Rabbit Lungs—PJ Coker, LA Hernandez, KJ Peevy, K Adkins, JC Parker. Crit Care Med 1992;20:635.

OBJECTIVES: To study the individual and combined effects of surfactant inactivation and mechanical ventilation on pulmonary microvascular permeability and lung compliance. DESIGN: Prospective, controlled trial. An isolated, perfused, lung model of surfactant inactivation and mechanical ventilation at 15, 30, and 45 cm H_2O peak inspiratory pressure was developed in young (4-6 wks) New Zealand white rabbits. SETTING: Laboratory of a university-affiliated medical school. MEASUREMENTS & MAIN RESULTS: Isolated, perfused lungs were prepared for measurement of the capillary filtration coefficient before and after 1 of 4 interventions: instillation of dioctyl succinate, a surfactant inactivator, without ventilation (Group 1); ventilation without dioctyl succinate at 15, 30, or 45 cm H_2O peak inspiratory pressure (Group 2); ventilation after dioctyl succinate pretreatment at 15, 30, or 45 cm H_2O peak inspiratory pressure (Group 3); and control lungs without dioctyl succinate or ventilation (Group 4). A significant increase in the capillary filtration coefficient was noted after dioctyl succinate treatment alone, after ventilation alone at 45 cm H_2O peak inspiratory pressure, and after dioctyl succinate plus ventilation at 15, 30, and 45 cm H_2O peak inspiratory pressure. Dioctyl succinate plus ventilation pro-

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INDICATIONS AND USAGE

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Prevention

In premature infants less than 1250 g birth weight or with evidence of surfactant deficiency, give SURVANTA as soon as possible, preferably within 15 minutes of birth.

Rescue

To treat infants with RDS confirmed by x-ray and requiring mechanical ventilation, give SURVANTA as soon as possible, preferably by 8 hours of age.

CONTRAINDICATIONS

None known.

WARNINGS

SURVANTA is intended for intratracheal use only.

SURVANTA CAN RAPIDLY AFFECT OXYGENATION AND LUNG COMPLIANCE. Therefore, its use should be restricted to a highly supervised clinical setting with immediate availability of clinicians experienced with intubation, ventilator management, and general care of premature infants. Infants receiving SURVANTA should be frequently monitored with arterial or transcutaneous measurement of systemic oxygen and carbon dioxide.

DURING THE DOSING PROCEDURE, TRANSIENT EPISODES OF BRADYCARDIA AND DECREASED OXYGEN SATURATION HAVE BEEN REPORTED. If these occur, stop the dosing procedure and initiate appropriate measures to alleviate the condition. After stabilization, resume the dosing procedure.

PRECAUTIONS

General

Rales and moist breath sounds can occur transiently after administration. Endotracheal suctioning or other remedial action is not necessary unless clear-cut signs of airway obstruction are present.

Increased probability of post-treatment nosocomial sepsis in SURVANTA-treated infants was observed in the controlled clinical trials (Table 3). The increased risk for sepsis among SURVANTA-treated infants was not associated with increased mortality among these infants. The causative organisms were similar in treated and control infants. There was no significant difference between groups in the rate of post-treatment infections other than sepsis.

Use of SURVANTA in infants less than 600 g birth weight or greater than 1750 g birth weight has not been evaluated in controlled trials. There is no controlled experience with use of SURVANTA in conjunction with experimental therapies for RDS (eg, high-frequency ventilation or extracorporeal membrane oxygenation).

No information is available on the effects of doses other than 100 mg phospholipids/kg, more than four doses, dosing more frequently than every 6 hours, or administration after 48 hours of age.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Reproduction studies in animals have not been completed. Mutagenicity studies were negative. Carcinogenicity studies have not been performed with SURVANTA.

ADVERSE REACTIONS

The most commonly reported adverse experiences were associated with the dosing procedure. In the multiple-dose controlled clinical trials, transient bradycardia occurred with 11.9% of doses. Oxygen desaturation occurred with 9.8% of doses.

Other reactions during the dosing procedure occurred with fewer than 1% of doses and included endotracheal tube reflux, pallor, vasoconstriction, hypotension, endotracheal tube blockage, hypertension, hypocarbia, hypercarbia, and apnea. No deaths occurred during the dosing procedure, and all reactions resolved with symptomatic treatment.

The occurrence of concurrent illnesses common in premature infants was evaluated in the controlled trials. The rates in all controlled studies are in Table 3.

TABLE 3

	All Controlled Studies		
Concurrent Event	SURVANTA (%)	Control (%)	*P* Value[a]
Patent ductus arteriosus	46.9	47.1	0.814
Intracranial hemorrhage	48.1	45.2	0.241
Severe intracranial hemorrhage	24.1	23.3	0.693
Pulmonary air leaks	10.9	24.7	< 0.001
Pulmonary interstitial emphysema	20.2	38.4	< 0.001
Necrotizing enterocolitis	6.1	5.3	0.427
Apnea	65.4	59.6	0.283
Severe apnea	46.1	42.5	0.114
Post-treatment sepsis	20.7	16.1	0.019
Post-treatment infection	10.2	9.1	0.345
Pulmonary hemorrhage	7.2	5.3	0.166

[a]*P*-value comparing groups in controlled studies

When all controlled studies were pooled, there was no difference in intracranial hemorrhage. However, in one of the single-dose rescue studies and one of the multiple-dose prevention studies, the rate of intracranial hemorrhage was significantly higher in SURVANTA patients than control patients (63.3% *v* 30.8%, $P = 0.001$, and 48.8% *v* 34.2%, $P = 0.047$, respectively). The rate in a Treatment IND involving approximately 4400 infants was lower than in the controlled trials.

In the controlled clinical trials, there was no effect of SURVANTA on results of common laboratory tests: white blood cell count and serum sodium, potassium, bilirubin, creatinine.

More than 3700 pretreatment and post-treatment serum samples were tested by Western Blot immunoassay for antibodies to surfactant-associated proteins SP-B and SP-C. No IgG or IgM antibodies were detected.

Several other complications are known to occur in premature infants. The following conditions were reported in the controlled clinical studies. The rates of the complications were not different in treated and control infants, and none of the complications were attributed to SURVANTA.

Respiratory: lung consolidation, blood from the endotracheal tube, deterioration after weaning, respiratory decompensation, subglottic stenosis, paralyzed diaphragm, respiratory failure.

Cardiovascular: hypotension, hypertension, tachycardia, ventricular tachycardia, aortic thrombosis, cardiac failure, cardiorespiratory arrest, increased apical pulse, persistent fetal circulation, air embolism, total anomalous pulmonary venous return.

Gastrointestinal: abdominal distention, hemorrhage, intestinal perforations, volvulus, bowel infarct, feeding intolerance, hepatic failure, stress ulcer.

Renal: renal failure, hematuria.

Hematologic: coagulopathy, thrombocytopenia, disseminated intravascular coagulation.

Central Nervous System: seizures.

Endocrine/Metabolic: adrenal hemorrhage, inappropriate ADH secretion, hyperphosphatemia.

Musculoskeletal: inguinal hernia.

Systemic: fever, deterioration.

Follow-Up Evaluations

To date, no long-term complications or sequelae of SURVANTA therapy have been found.

Single-Dose Studies

Six-month adjusted-age follow-up evaluations of 232 infants (115 treated) demonstrated no clinically important differences between treatment groups in pulmonary and neurologic sequelae, incidence or severity of retinopathy of prematurity, rehospitalizations, growth, or allergic manifestations.

Multiple-Dose Studies

Six-month adjusted age follow-up evaluations have not been completed. Preliminarily, in 605 (333 treated) of 916 surviving infants, there are trends for decreased cerebral palsy and need for supplemental oxygen in SURVANTA infants. Wheezing at the time of examination tended to be more frequent among SURVANTA infants, although there was no difference in bronchodilator therapy.

Twelve-month follow-up data from the multiple-dose studies have been completed in 328 (171 treated) of 909 surviving infants. To date no significant differences between treatments have been found, although there is a trend toward less wheezing in SURVANTA infants in contrast to the six month results.

OVERDOSAGE

Overdosage with SURVANTA has not been reported. Based on animal data, overdosage might result in acute airway obstruction. Treatment should be symptomatic and supportive.

Rales and moist breath sounds can transiently occur after SURVANTA is given, and do not indicate overdosage. Endotracheal suctioning or other remedial action is not required unless clear-cut signs of airway obstruction are present.

HOW SUPPLIED

SURVANTA (beractant) Intratracheal Suspension is supplied in single-use glass vials containing 8 mL of SURVANTA (NDC 0074-1040-08). Each milliliter contains 25 mg of phospholipids (200 mg phospholipids/8 mL) suspended in 0.9% sodium chloride solution. The color is off-white to light brown.

Store unopened vials at refrigeration temperature (2-8°C). Protect from light. Store vials in carton until ready for use. Vials are for single use only. Upon opening, discard unused drug.

June, 1991

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duced a significantly greater increase in the capillary filtration coefficient than ventilation alone at 15 and 45 cm H_2O peak inspiratory pressure. CONCLUSIONS: These data suggest that ventilation after surfactant inactivation is more injurious to the pulmonary microvasculature than ventilation alone, and that generalized lung overdistention is not the primary mechanism for microvascular injury in the diseased, noncompliant lung. The increases seen in the capillary filtration coefficient in postventilated surfactant inactivated lungs, even at low-ventilation pressures, suggest that low peak inspiratory pressures do not overdistend the dioctyl succinate-treated lung.

Dissociation of Symptom Scores and Bronchial Hyperreactivity: Study in Asthmatic Children on Long-Term Treatment with Inhaled Beclomethasone Dipropionate—MR Benoist, JJ Brouard, P Rufin, S Waernessyckle, J de Blic, J Paupe, P Scheinmann. Pediatr Pulmonol 1992;13:71.

SUMMARY: The aim of the study was to evaluate the effects of inhaled steroids (IS) on the improvement of clinical asthma symptoms and on the decrease in bronchial hyperreactivity (BHR). Twenty-four children with severe asthma were given 1,000 μg beclomethasone dipropionate (BDP) daily and compared with 10 asthmatic control children. The study included the evaluation of daily clinical score, of exercise-induced asthma, of bronchial obstruction (forced expiratory volume in 1 second, FEV_1), and of BHR at months 0, 1, 2-3, and 4-5 (M0, M1, M2-3, and M4-5). BHR was assessed by standardized inhaled carbachol provocation measuring plethysmographic specific airway resistance (sR_{aw}). The carbachol dose causing a 40% decrease in specific conductance (sG_{aw}) was determined (PD_{40} sG_{aw}). Clinical scores decreased at M1 ($p < 0.01$) and throughout the study. FEV_1 increased at M1 ($p < 0.05$), M2-3 ($p < 0.01$), and M4-5 ($p < 0.05$) compared to M0. PD_{40} sG_{aw} only increased significantly at M1 and M2-3. No individual correlation was found between clinical scores and PD_{40} sG_{aw} at any testing, or between the decrease of clinical scores and the decrease of BHR. We conclude that bronchoconstrictive challenge tests do not adequately assess the clinical efficacy of IS. In clinical practice non-specific BHR should be preferentially measured for diagnosing atypical forms of asthma.

A Simple Method for the Measurement of Intrinsic Positive End-Expiratory Pressure during Controlled and Assisted Modes of Mechanical Ventilation—SB Gottfried, E Reissman, VM Ranieri. Crit Care Med 1992;20:621.

OBJECTIVE: To evaluate a new and simple method for the measurement of intrinsic positive end-expiratory pressure during controlled and assisted modes of mechanical ventilation. DESIGN: Prospective study. SETTING: 3 university hospital medical ICUs. PATIENTS: A total of 13 intubated, mechanically ventilated patients with severe airway obstruction. INTERVENTIONS: Airway occlusions reproducibly timed to occur coincidently with end-expiration were obtained by (a) manipulation of a three-way manual valve placed in the inspiratory limb of the external ventilator circuit (manual valve method) and (b) activation of the expiratory pause hold function of the mechanical ventilator (Siemens 900C). MEASUREMENTS & MAIN RESULTS: Airway pressure, flow, and volume were recorded during controlled and assisted modes of mechanical ventilation. Intrinsic positive end-expiratory pressure was determined from the plateau in airway pressure, which was developed during end-expiratory occlusions. For controlled mechanical ventilation, intrinsic positive end-expiratory pressure averaged 11.42 ± 0.77 (SEM) cm H_2O with the manual valve method, compared with 11.38 ± 0.70 cm H_2O, using the ventilator expiratory pause hold function. There was close correlation between results over the wide range of intrinsic positive end-expiratory pressure observed, which varied from approximately 5 to 22 cm H_2O ($y = 1.08\times - 0.92$; $r^2 = 0.99$). Values of intrinsic positive end-expiratory pressure were comparable for the two methods during assist-control ventilation, pressure support ventilation, and spontaneous breathing through the ventilator circuit. The manual valve method was also effective when tested with different mechanical ventilators using a mechanical lung model. CONCLUSIONS: The manual valve method can be used to determine intrinsic positive end-expiratory pressure during controlled and assisted modes of ventilatory support with current ventilators. The availability of such an approach should facilitate the routine monitoring of intrinsic positive end-expiratory pressure in mechanically ventilated patients, thereby aiding clinical decision making and management in these critically ill individuals.

Relative Humidity, Not Absolute Humidity, Is of Great Importance When Using a Humidifier with a Heating Wire—H Miyao, T Hirokawa, K Miyasaka, T Kawazoe. Crit Care Med 1992;20:674.

OBJECTIVES: Since the introduction of a humidifier with a heating wire, we have frequently experienced severe upper airway obstruction from consolidation of secretions, previously unencountered when a humidifier without a heating wire

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was used. Such problems led to the suspicion that the heating wire incorporated into the breathing circuit of the heated humidifier might be the cause. Therefore, we scheduled an experiment to assess the hypothesis that relative humidity, rather than absolute humidity, is a dominant factor in the case of drying secretions in the upper airway when using such a humidifier. DESIGN: 3 clinical case reports and an experiment with a tracheal model. SETTING: Intensive care units at Saitama Medical Center, Saitama Medical School, Saitama, Japan. PATIENTS: 3 intubated patients. MEASUREMENTS & MAIN RESULTS: An experiment with a tracheal model showed that gas with a higher temperature and lower relative humidity (35°C, 48%) deprived the tracheal model of significantly more water (5.9 ± 0.21 [SD] g) than gas with a lower temperature and higher relative humidity (24°C, 87%) (2.9 ± 0.4 g; $p < 0.01$), even though the gases contained the same amount of water vapor (19 mg H_2O/L) minus the same absolute humidity. CONCLUSIONS: A heated humidifier with a heating wire incorporated into the breathing circuit may be dangerous when only temperature is monitored and controlled. Relative humidity, rather than absolute humidity, is a dominant factor in the case of drying secretions in the upper airway when using such a humidifier.

Positional Oxygenation Changes in Air-Transported Neonates—SJ Squire, KT Kirchhoff. Heart Lung 1992;21: 255.

The effect of position on oxygenation was compared in 24 critically ill, premature neonates during pressurized, fixed-wing transport. The purpose of the study was to assess if there is a significant change in the arterial oxygen tension in the supine, prone, or prone-free positions during air transport. Neonates with

endotracheal tubes in place who (1) had radiologic evidence of hyaline membrane disease, (2) had an umbilical artery catheter in place, and (3) required at least 1 hour of air transport to a Level III intensive care center were admitted into the study after parental consent. There was an average 8% increase in oxygenation in the prone and prone-free positions when compared with the supine position. Although this difference might be considered clinically significant for the critically ill premature neonate, there was no statistically significant difference in arterial oxygen tension among the three positions tested in this study.

Cardiorespiratory Effects of Changing Inspiratory to Expiratory Ratio during High-Frequency Oscillation in an Animal Model of Respiratory Failure—SE Courtney, KR Weber, WA Spohn, CV Bender, SW Malin, RW Gotshall. Pediatr Pulmonol 1992;13:113.

SUMMARY: To examine the effects of varying inspiratory-expiratory ratio (I:E) on cardiorespiratory function during high-frequency oscillation (HFO), 11 saline-lavaged rabbits were ventilated at I:E = 1:2, 1:1.5, 1.5:1, and 2:1 in a paired comparison to a baseline of I:E = 1:1. HFO was delivered by a SensorMedics Model 3100 Oscillator at a frequency of 10 Hz. Pressure amplitude and proximal mean airway pressure ($D\bar{P}_{aw}$) were held constant as I:E was varied from baseline to the experimental I:E. During each paired observation, P_{aO_2}, P_{aCO_2}, cardiac output, blood pressure, and distal mean airway pressure ($D\bar{P}_{aw}$) were measured. We found that as I:E was increased or decreased from 1:1, no significant changes in P_{aO_2}, P_{aCO_2}, blood pressure, or cardiac output occurred. We conclude that in this model, varying I:E has no significant effect on oxygenation, ventilation, or cardiovascular function.

Effects of Diltiazem on Oxygen Delivery and Consumption after Asphyxial Cardiac Arrest and Resuscitation—KH Lindner, AW Prengel, FW Ahnefeld, H Ensinger, W Schürmann. Crit Care Med 1992;20:650.

BACKGROUND & METHODS: Calcium-channel blockers may attenuate vasospasm after transient ischemia and improve organ blood flow after resuscitation. Our aim was to assess the effect of diltiazem on systemic oxygen delivery and consumption, hemodynamics, electroencephalogram (EEG), and organ blood flow after restoration of spontaneous circulation. After a 3-min period of asphyxial cardiac arrest, 14 pigs (20-27 kg) were randomly allocated to treatment with either diltiazem (0.1 mg/kg bolus followed by an I.V. infusion of 0.025 mg/min/kg over 120 mins) or placebo, given at 5 mins after successful resuscitation. Organ blood flow was measured using tracer microspheres 120 mins after resumption of spontaneous circulation. RESULTS: Median systemic oxygen delivery index values at 30, 60, and 120 mins after restoration of spontaneous circulation were 18.2 mL/min/kg (range 14.8-20.7), 16.8 mL/min/kg (13.2-20.8), and 19.6 mL/min/kg (16.9-21.0), respectively, in the diltiazem group and 13.1 mL/min/kg (11.2-14.6), 11.9 mL/min/kg (10.3-13.3), and 14.7 mL/min/kg (11.4-17.2), respectively, in the control group ($p < 0.05$ for all three comparisons). At the same points in time, median systemic oxygen consumption indices were 3.2 mL/min/kg (range 2.2-3.7), 2.1 mL/min/kg (1.9-3.0), and 2.6 mL/min/kg (1.8-3.8) in the diltiazem group and 2.8 mL/min/kg (2.1-4.0), 2.7 mL/min/kg (1.7-4.3), and 2.3 mL/min/kg (1.6-3.8) in the placebo group (NS). Diltiazem enhanced the postarrest recovery of EEG total power. Right and left cerebral blood flow 120 mins after restoration of spontaneous circulation was significantly ($p < 0.01$) higher in the diltiazem group in comparison with the control group. CONCLUSIONS: Diltiazem causes an increase in systemic oxygen delivery index by promoting vasodilation, but it does not change systemic oxygen consumption index in comparison to placebo treatment. It may be that an impairment in local autoregulation and/or in oxidative metabolism at the cellular or subcellular level was the reason why diltiazem did not improve these derangements. The observed increase in cerebral blood flow and in EEG recovery may be beneficial to the brain after a period of asphyxia.

Attitudes of Medical Students, Housestaff, and Faculty Physicians toward Euthanasia and Termination of Life-Sustaining Treatment—PV Caralis, JS Hammond. Crit Care Med 1992;20:683.

OBJECTIVES: Medical decisions concerning the prolongation of life, the right to die, and euthanasia are among the most extensively discussed decisions within medicine and law today. The responses of 360 physicians, housestaff, and medical students to a questionnaire were analyzed to identify attitudes toward these issues. DESIGN: Case vignettes were utilized to simulate the clinical context within which to survey decisions regarding whether or not to allow and assist patients requesting to die. MEASUREMENTS & MAIN RESULTS: The majority of respondents (76%) consider withholding and withdrawing life-support therapy consistent with passive euthanasia. Passive euthanasia is more acceptable to the majority of the respondents (77%) and all three groups (physicians, housestaff, and students) are similarly more disturbed by active euthanasia. Of all respondents, 51% would accede to the patient's

wishes when life-saving treatment is refused, but only 16% would do so when a patient requested assistance in dying. Despite the fact that a majority (68%) agree that there is a moral justification for assisting patients to die and feel "understanding" for a physician who assists a patient in dying, only 6% of those persons surveyed were willing to deliberately terminate the life of a patient by administering medication to cause respiratory arrest, and only 1.1% of those persons surveyed were willing to do so to cause cardiac arrest. In the case vignettes, the faculty placed their highest value on disease-based information as strongly determinative to their decisions, while students and housestaff preferred quality-of-life factors. Respondents uniformly found it easier to perform "passive" actions; they were more willing to perform "active" actions in case vignettes where patients had terminal illnesses. CONCLUSIONS: Socially and legally created "shades of gray" have blurred the distinctions between withholding or withdrawing therapies and euthanasia and have left physicians without guidelines. Health ethics education should focus on case-based teaching and on reducing the uncertainty at the bedside.

The Effects of Airway Hyperresponsiveness, Wheezing, and Atopy on Longitudinal Pulmonary Function in Children: A 6-Year Follow-Up Study—D Sherrill, MR Sears, MD Lebowitz, D Holdaway, CJ Hewitt, EM Flannery, et al. Pediatr Pulmonol 1992;13:78.

SUMMARY: We examined growth of spirometric lung function in 696 children of European ancestry who were followed from ages 9 to 15 years and stratified according to their degree of responsiveness to methacholine inhalation challenge, atopic status, and respiratory symptoms. Subjects were participants in the longitudinal Multidisciplinary Health and Development Study in Dunedin, New Zealand. Forced expired volume in 1 second (FEV_1) and vital capacity (VC) were measured at 9, 11, 13, and 15 years of age, concurrently with assessment of airway responsiveness determined by the concentration of methacholine causing a 20% fall in FEV_1 (PC_{20} FEV_1). Atopic status was assessed at age 13 by skin-prick testing to 11 allergens. In children demonstrating airway hyperresponsiveness, FEV_1 increased with age at a slower rate, and the FEV_1/VC ratio had a faster rate of decline through childhood, compared to non-responsive children. Subjects with positive skin tests to house dust mite and cat dander also had lower mean FEV_1/VC ratios than the control group. Any reported wheezing was associated with slower growth of FEV_1 and VC in males. We conclude that in New Zealand children with airway responsiveness and/or atopy to house dust mite or cat growth of spirometric lung function is impaired.

Flow-Volume Relationships during Spontaneous Breathing through Endotracheal Tubes—C Weissman. Crit Care Med 1992;20:615.

OBJECTIVE: Breathing spontaneously through an endotracheal tube is often equated with "breathing through a straw." The objective of this study was to examine whether endotracheal tubes functionally act as tubular resistors in patients during tidal breathing, machine-initiated breaths, and vital capacity maneuvers. DESIGN: Descriptive study. SETTING: University hospital surgical ICU. PATIENTS: 18 mechanically ventilated, critically ill, postoperative patients (34-87 yrs). MEASUREMENTS: Flow-volume loops were recorded during tidal and machine breaths, as well as during vital capacity maneuvers. The loops were inspected visually for evidence of airflow limitation. Expiratory characteristics were examined using the ratio of expiratory volume in 1 second to the expiratory volume in 0.5 second and the ratio of expiratory volume in 1 second to peak expiratory flowrate. The ratio of expiratory volume in 1 second to that in 0.5 second (> 1.5) and the ratio of expiratory volume in 1 second to peak expiratory flowrate (> 10 $mL \cdot min \cdot L^{-1}$) are seen with upper airway obstruction. MAIN RESULTS: Mean (SD) spontaneous tidal volume was 361 (92) mL and vital capacity 1104 (16) mL, while the ratio of expiratory volume in 1 second to that in 0.5 second was 1.57 (0.17) and 1.7 (0.1) ($p < 0.05$), respectively. The ratio of expiratory volume in 1 second to peak expiratory flowrate was 9.9 (1.7) during tidal breathing and 12.9 (1.2) ($p < 0.05$) during vital capacity maneuvers. CONCLUSIONS: Endotracheal tubes of the diameters used in these adult postoperative patients (7 and 8 mm) cause only minimal limitation to airflow during the small volumes and low flowrates observed during tidal breathing. Evidence of greater airflow limitation was seen during vital capacity maneuvers.

High-Frequency Jet Ventilation and Surfactant Treatment of Newborns with Severe Respiratory Failure—JM Davis, SE Richter, JW Kendig, RH Notter. Pediatr Pulmonol 1992;13:108.

SUMMARY: 28 newborn infants (birthweight 2.4 ± (1.1 kg; gestational age 34.6 ± 6.1 weeks) with respiratory distress syndrome (RDS), meconium aspiration syndrome, or pneumonia who deteriorated in spite of optimal conventional mechanical ventilation (CMV) and exogenous surfactant therapy were treated with high-frequency jet ventilation (HFJV) and continued surfactant therapy. For

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enrollment, infants had to have a limited response to surfactant therapy and conventional ventilation, and meet clinical criteria that confirmed clinical deterioration and severity of illness. Study infants had received exogenous calf lung surfactant extract (CLSE) and conventional ventilation prior to the start of HFJV at 46.3 ± 8.2 hours of age. Patients initially responded to HFJV alone with significant improvement in several respiratory variables, but deteriorated subsequently and received additional doses of exogenous surfactant on HFJV. Exogenous surfactant and HFJV resulted in significant and sustained improvement in several respiratory variables. Only 10 patients deteriorated to meet criteria for a second surfactant dose on HFJV, and 2 patients received a third dose. Twenty-five of the 28 patients studied survived (89%). No patients received extracorporeal membrane oxygenation or were discharged home on oxygen. The results of this pilot study suggest that the combination of HFJV and exogenous surfactant replacement may be effective in treating infants with more severe respiratory failure, and indicate the need for more extensive controlled investigations.

Lack of Relationship between the True Airway Pressure and the Pressure Displayed with an Infant Ventilator—A Sola, D Farina, S Rodriguez, I Kurlat. Crit Care Med 1992;20:778.

OBJECTIVE: To establish if the pressure indicated in the manometer of an infant ventilator (IV 100B, Sechrist, Anaheim CA) reflects the true pressure delivered to the proximal airway during mechanical ventilation in the neonatal ICU. DESIGN: With approval of our Institutional Research Board, data were collected prospectively. Peak inspiratory pressure and end-expiratory pressure were measured at the 'Y' piece of the breathing tubing. Pressure readings from the conventional ventilator's manometer were compared with simultaneously obtained measurements using an electronic monitor. SETTING: This study was conducted in a 45-bed neonatal ICU, admitting 700 to 750 newborns per year. PATIENTS: 12 neonates who required mechanical ventilation were included in the study. INTERVENTIONS: Specific interventions were not made by study design. Measurements routinely obtained were compared. MEASUREMENTS & MAIN RESULTS: 275 simultaneous measurements of peak inspiratory pressure and positive end-expiratory pressure were compared. Peak inspiratory pressure values were higher with the electronic monitor in 273 (99%) of 275 measurements and the mean of the differences between the electronic monitor and ventilator's manometer was statistically significant ($p < 0.001$). For positive end-expiratory pressure measurements, values indicated by the electronic monitor were lower in 152 (55%) of 275 determinations, equal in 65 (23%), and higher in 58 (21%) determinations. Percent variations between methods ranged from 0-140% for peak inspiratory pressures and from 0-500% for positive end-expiratory pressure. CONCLUSIONS: These data demonstrate that it is impossible to know the true pressure delivered to the proximal airway of a neonate during mechanical ventilation by observing the ventilator pressure manometer. The manometer readings consistently underestimate the true peak inspiratory pressure values and are very unpredictable regarding positive end-expiratory pressure values. These findings support the use of other methods to monitor the proximal airway pressure besides the ventilator's manometer in the neonatal ICU. Furthermore, mean airway pressure should not be calculated from the pressure readings obtained from the tested ventilator's manometer.

Pediatric Asthma Deaths in Victoria: The Mild Are At Risk—CF Robertson, AR Rubinfeld, G Bowes. Pediatr Pulmonol 1992;13:95.

SUMMARY: Previous reviews of pediatric asthma mortality have mostly been from hospital-based clinic populations and suggest that only those with severe asthma are most at risk. This report summarizes an investigation, by interviewer-administered questionnaire, into the circumstances surrounding the death in all patients aged 20 years or less who died from asthma in the State of Victoria over a 3-year period from May 1, 1986. During this period, 51 deaths due to asthma were reported. Thirty-three percent of these were judged to have a history of trivial or mild asthma, and 32% had no previous hospital admission for asthma. However, 36% were judged to have had severe asthma, 43% were taking regular inhaled beclomethasone or sodium cromoglycate, and 10% were taking regular oral steroids. Twenty-two percent had a previous admission to an ICU. Death occurred outside hospital in 40 (78%) subjects. In the final attack 63% had sudden onset and collapse within minutes, 12% were found dead, and 25% had acute progression of an established attack. The investigators assessed 39% of the deaths to have had potentially preventable elements. The preventable factors included inadequate assessment or therapy of prior asthma (68%), poor compliance with therapy (53%), and delay in seeking help (47%). The majority of subjects in this survey could not be classified as "high risk." Therefore, clinicians should ensure that all young patients with asthma are aware of optimal maintenance management, can recognize deteriorating asthma, and follow a clear individualized crisis plan.

Indirect Calorimetry in Mechanically Ventilated Infants and Children: Measurement Accuracy with Absence of Audible Airleak—WJ Chwals, KP Lally, MM Woolley. Crit Care Med 1992;20:768.

OBJECTIVE: To establish the effect of an audible airleak (around an endotracheal tube) on oxygen consumption ($\dot{V}_{O_2}$) measurements in pediatric ICU patients. DESIGN: Prospective trial comparing $\dot{V}_{O_2}$ measurements before and after deflation of the endotracheal tube cuff. SETTING: Pediatric ICU in a large pediatric tertiary care center. PATIENTS: 20 critically ill infants and children receiving mechanical ventilatory support via cuffed endotracheal tube. INTERVENTIONS: Deflation of endotracheal tube cuff. MEASUREMENTS & MAIN RESULTS: The presence (Group 1, n = 9) or absence (Group 2, n = 11) of an audible airleak with the cuff deflated was confirmed by two independent observers. The percent difference in $\dot{V}_{O_2}$ was calculated for both groups using the following formula: ([$\dot{V}_{O_2}$ cuff up $-\dot{V}_{O_2}$cuff down]/$\dot{V}_{O_2}$cuff up) x 100. An audible airleak associated with cuff deflation (Group 1) caused a significant ($p = 0.0012$) reduction of $\dot{V}_{O_2}$ by 45.6% (mean difference in $\dot{V}_{O_2}$ = 45.6%). In contrast, with no audible airleak after cuff deflation (Group 2), only minimal changes in $\dot{V}_{O_2}$ (mean difference in $\dot{V}_{O_2} = -0.4\%$) were observed. CONCLUSIONS: These data suggest that if no audible airleak is detected, $\dot{V}_{O_2}$ determined by indirect calorimetry may be reliably measured in infants and children with a noncuffed endotracheal tube.

Lung Mechanics during and after Extracorporeal Membrane Oxygenation for Meconium Aspiration Syndrome—AC Koumbourlis, EK Motoyama, RL Mutich, DK Nakayama, AE Thompson. Crit Care Med 1992;20:751.

OBJECTIVE: To determine whether abnormalities in lung mechanics detected in infants during the acute phase of meconium aspiration syndrome persist after treatment with extracorporeal membrane oxygenation (ECMO). DESIGN: Prospective, descriptive study. Prospective evaluation of airway function and lung mechanics during and after ECMO by pulmonary function testing at 1.8 ± 0.5 days of EMCO (Period 1), follow-up at 1.4 ± 0.2 days (Period 2), and 7.0 ± 0.9 days (Period 3) after decannulation from ECMO. SETTING: Tertiary care neonatal/pediatric ICU. PATIENTS: 12 neonates undergoing ECMO treatment for severe meconium aspiration syndrome that was refractory to conventional mechanical ventilation. INTERVENTIONS: Maximum expiratory flow-volume curves were studied with the deflation flow-volume curve technique, and compliance and resistance of the respiratory system were studied with partial passive flow-volume curves. MEASUREMENTS & MAIN RESULTS: Respiratory system compliance was the only index of respiratory mechanics that was significantly ($p < 0.05$) improved (0.96 ± 0.1 vs 0.61 ± 0.1 mL · kg · cm H_2O^{-1}) immediately after decannulation from ECMO compared with Period 1. Clinically important ($p < 0.05$) improvement in forced vital capacity (28.0 ± 5.5 vs 16.1 ± 1.9 mL/kg), respiratory system compliance (1.01 ± 0.2 vs 0.61 ± 0.1 mL · kg · cm H_2O^{-1}), and maximum expiratory flow at 25%/forced vital capacity (1.0 ± 0.3 vs 2.2 ± 0.3) was evident only during Period 3 compared with Period 1. CONCLUSIONS: We conclude that improvements in the clinical condition and oxygenation, permitting successful decannulation from ECMO, are achieved before clinically important improvements in lung mechanics.

Colonization of Dental Plaque by Respiratory Pathogens in Medical Intensive Care Patients—FA Scannapieco, EM Stewart, JM Mylotte. Crit Care Med 1992;20:740.

OBJECTIVE: To assess the prevalence of oral colonization by respiratory pathogens in a group of ICU patients, with specific attention to dental plaque and the oral mucosa. DESIGN: Prospective, nonrandomized study with age-matched controls. SETTINGS: Medical ICU in a tertiary-care Veterans Affairs Medical Center and a dental school outpatient preventive dentistry clinic. PATIENTS: Nonconsecutive, unselected patients admitted to the medical ICU during a 2-mo period; controls were age-matched patients seen for the first time in the preventive dentistry clinic. INTERVENTIONS: None. MEASUREMENTS: Oral hygienic status was assessed in both groups using a semiquantitative system. Quantitative cultures of dental plaque and buccal mucosa were done within 12 hours of medical ICU admission and every third day thereafter until discharge/death from the medical ICU. In controls, cultures of plaque and buccal mucosa were done on the initial visit only. Severity of illness of medical ICU patients was quantitated using the Acute Physiology and Chronic Health Evaluation (APACHE II) system and McCabe-Jackson criteria. MAIN RESULTS: Oral hygiene of medical ICU patients was poor. These patients had a mean plaque score (1.9 ± 0.2) that was significantly greater than that same score seen in outpatients of the preventive dentistry clinic (1.4 ± 0.1; $p < 0.005$). Plaque and/or oral mucosa of 22 (65%) of 34 medical ICU patients were colonized by respiratory pathogens, in contrast to only 4 (16%) of 25 preventive dentistry clinic patients ($p < 0.005$). The potential respiratory pathogens cultured from medical ICU patients in-

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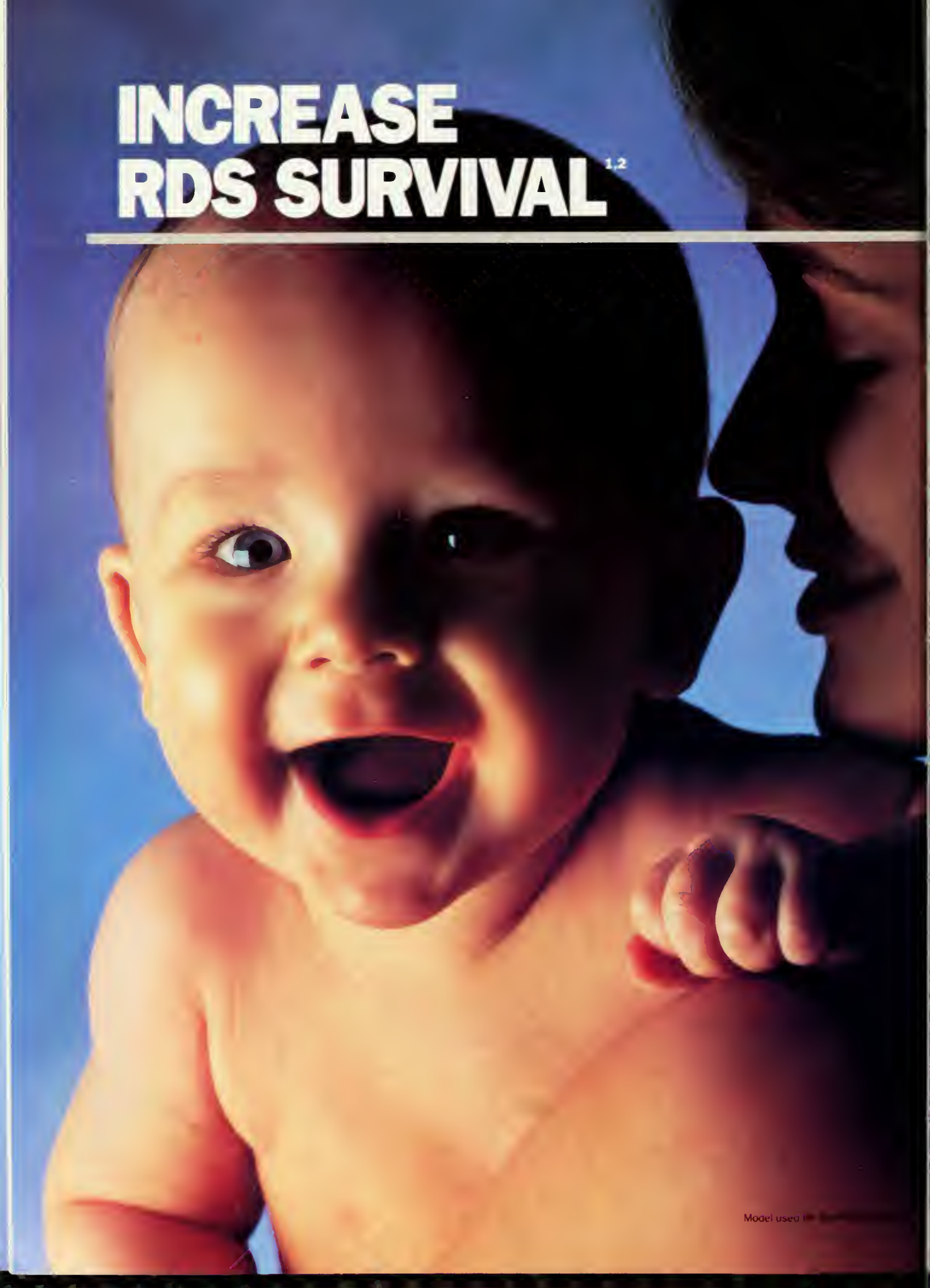

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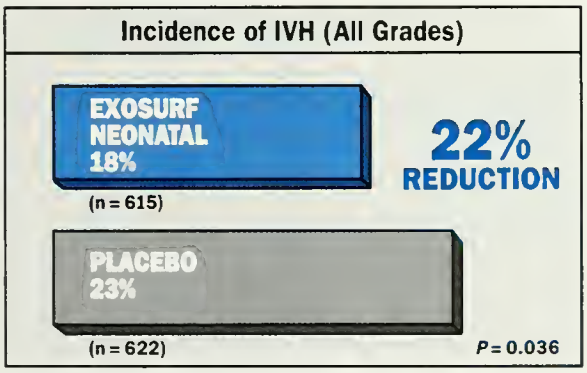

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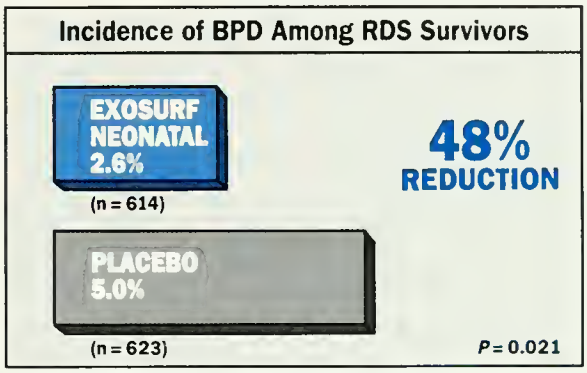
Adapted from Long et al[2 (p1700)]

Significant BPD Reduction in Infants ≥1250 g

In the rescue trial involving over 1200 infants, administration of EXOSURF Neonatal significantly reduced the incidence of bronchopulmonary dysplasia (BPD) by nearly 50% ($P = 0.021$).



Two-dose treatment. Incidence of BPD in 28-day survivors following RDS in infants weighing 1250 g or more.
Adapted from Long et al[2 (p1699)]

No Increase in Sepsis

No difference in the incidence of sepsis has been seen with EXOSURF Neonatal during placebo-controlled trials (n = 1517). The rate of sepsis was similar in an open trial of 11,455 infants.[6]

No Animal Proteins

EXOSURF is purely synthetic and carries no known infectious or immunologic risks.

Other Safety Considerations

Various forms of pulmonary air leak were reduced in all controlled trials. A single controlled study in infants 500-699 g reported a significant increase in pulmonary hemorrhage.[7] Significant increases in apnea were reported in three controlled trials.[1,2,8] Apnea appears to be a marker for improved survival.[2]

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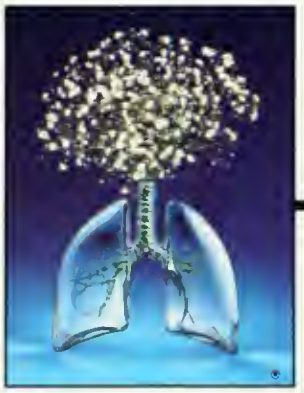
INCREASES RDS SURVIVAL... REDUCES RISKS*

Please see brief summary of full prescribing information on following page.

*Increased pulmonary hemorrhage was noted in one trial of infants 500-699 g; increased apnea has been noted in some trials.[1,2,8]



Exosurf® NEONATAL™
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*Increased pulmonary hemorrhage was noted in one trial of infants 500-699 g; increased apnea has been noted in some trials.[1,2,8]

PLEASE CONSULT FULL PRODUCT INFORMATION BEFORE PRESCRIBING

INDICATIONS AND USAGE: Exosurf Neonatal is indicated for: 1. **Prophylactic** treatment of infants with birth weights of less than 1350 grams who are at risk of developing RDS (see PRECAUTIONS). 2. **Prophylactic** treatment of infants with birth weights greater than 1350 grams who have evidence of pulmonary immaturity, and 3. **Rescue** treatment of infants who have developed RDS.

CONTRAINDICATIONS: There are no known contraindications to treatment with Exosurf Neonatal.

WARNINGS: Intratracheal Administration Only: Exosurf Neonatal should be administered only by instillation into the trachea (see DOSAGE AND ADMINISTRATION). **General:** The use of Exosurf Neonatal requires expert clinical care by experienced neonatologists and other clinicians who are accomplished at neonatal intubation and ventilatory management. Adequate personnel, facilities, equipment, and medications are required to optimize perinatal outcome in premature infants. Vigilant clinical attention should be given to all infants prior to, during, and after administration of Exosurf Neonatal. **Acute Effects:** Exosurf Neonatal can rapidly affect oxygenation and lung compliance. **Lung Compliance:** If chest expansion improves substantially after dosing, peak ventilator inspiratory pressures should be reduced immediately, without waiting for confirmation of respiratory improvement by blood gas assessment. Failure to reduce inspiratory ventilator pressures rapidly in such instances can result in lung overdistention and fatal pulmonary air leak. **Hyperoxia:** If the infant becomes pink and transcutaneous oxygen saturation is in excess of 95%, FiO_2 should be reduced in small but repeated steps (until saturation is 90 to 95%) without waiting for confirmation of elevated arterial pO_2 by blood gas assessment. Failure to reduce FiO_2 in such instances can result in hyperoxia. **Hypocarbia:** If arterial or transcutaneous CO_2 measurements are <30 torr, the ventilator rate should be reduced at once. Failure to reduce ventilator rates in such instances can result in marked hypocarbia, which is known to reduce brain blood flow. **Pulmonary Hemorrhage:** In the single study conducted in infants weighing <700 grams at birth, the incidence of pulmonary hemorrhage (10% vs 2% in the placebo group) was significantly increased in the Exosurf Neonatal group. None of the five studies involving infants with birth weights >700 grams showed a significant increase in pulmonary hemorrhage in the Exosurf Neonatal group. In a cross-study analysis of these five studies, fatal pulmonary hemorrhage occurred in three infants; two in the Exosurf Neonatal group and one in the placebo group. Mortality from all causes among infants who developed pulmonary hemorrhage was 43% in the placebo group and 37% in the Exosurf Neonatal group. Pulmonary hemorrhage in both Exosurf Neonatal and placebo infants was more frequent in infants who were younger, smaller, male, or who had a patent ductus arteriosus. Pulmonary hemorrhage typically occurred in the first 2 days of life in both treatment groups. **Mucous Plugs:** Infants whose ventilation becomes markedly impaired during or shortly after dosing may have mucous plugging of the endotracheal tube, particularly if pulmonary secretions were prominent prior to drug administration. Suctioning of all infants prior to dosing may lessen the chance of mucous plugs obstructing the endotracheal tube. If endotracheal tube obstruction from such plugs is suspected, and suctioning is unsuccessful in removing the obstruction, the blocked endotracheal tube should be replaced immediately.

PRECAUTIONS: General: In the controlled clinical studies, infants known prenatally or postnatally to have major congenital anomalies, or who were suspected of having congenital infection, were excluded from entry. However, these disorders cannot be recognized early in life in all cases, and a few infants with these conditions were entered. The benefits of Exosurf Neonatal in the affected infants who received drug appeared to be similar to the benefits observed in infants without anomalies or occult infection. **Prophylactic Treatment—Infants <700 Grams:** In infants weighing 500 to 700 grams, a single prophylactic dose of Exosurf Neonatal significantly improved FiO_2 and ventilator settings, reduced pneumothorax and reduced death from RDS, but increased pulmonary hemorrhage (see WARNINGS). Overall mortality did not differ significantly between the placebo and Exosurf Neonatal groups. Data on multiple doses in infants in this weight class are not yet available. **Rescue Treatment—Number of Doses:** A small number of infants with RDS have received more than two doses of Exosurf Neonatal as rescue treatment. Definitive data on the safety and efficacy of these additional doses are not available. **Carcinogenesis, Mutagenesis, Impairment of Fertility:** Exosurf Neonatal at concentrations up to 10,000 µg/plate was not mutagenic in the Ames Salmonella assay. Long-term studies have not been performed in animals to evaluate the carcinogenic potential of Exosurf Neonatal. The effects of Exosurf Neonatal on fertility have not been studied.

ADVERSE REACTIONS:
General: Premature birth is associated with a high incidence of morbidity and mortality. Despite significant reductions in overall mortality associated with Exosurf Neonatal, some infants who received Exosurf Neonatal developed severe complications and either survived with permanent handicaps or died. In controlled clinical studies evaluating the safety and efficacy of Exosurf Neonatal, numerous safety assessments were made. In infants receiving Exosurf Neonatal, pulmonary hemorrhage, apnea and use of methylxanthines were increased. A number of other adverse events were significantly reduced in the Exosurf Neonatal group, particularly various forms of pulmonary air leak and use of pancuronium. ***Reflux:*** Reflux of Exosurf Neonatal into the endotracheal tube during dosing has been observed and may be associated with rapid drug administration. If reflux occurs, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O until the endotracheal tube clears. ***>20% Drop in Transcutaneous Oxygen Saturation:*** If transcutaneous oxygen saturation declines during dosing, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O for 1 to 2 minutes. In addition, increases of FiO_2 may be required for 1 to 2 minutes.

DOSAGE AND ADMINISTRATION: Preparation of Suspension: Exosurf Neonatal is best reconstituted immediately before use because it does not contain antibacterial preservatives. However, the reconstituted suspension is chemically and physically stable when stored at 2° to 30°C (36° to 86°F) for up to 12 hours following reconstitution. Solutions containing buffers or preservatives should not be used for reconstitution. **Do Not Use Bacteriostatic Water for Injection, USP.** Each vial of Exosurf Neonatal should be reconstituted only with **8 mL** of the accompanying diluent (preservative-free Sterile Water for Injection). **Dosage: Accurate determination of weight at birth is the key to accurate dosing.** ***Prophylactic Treatment:*** The first dose of Exosurf Neonatal should be administered as a single 5 mL/kg dose as soon as possible after birth. Second and third doses should be administered approximately 12 and 24 hours later to all infants who remain on mechanical ventilation at those times. ***Rescue Treatment:*** Exosurf Neonatal should be administered in two 5 mL/kg doses. The initial dose should be administered as soon as possible after the diagnosis of RDS is confirmed. The second dose should be administered approximately 12 hours following the first dose, provided the infant remains on mechanical ventilation. **Use of Special Endotracheal Tube Adapter:** With each vial of Exosurf Neonatal for Intratracheal Suspension, five different sized endotracheal tube adapters each with a special right angle Luer®-lock sideport are supplied. The adapters are clean but not sterile. **Administration:** The infant should be suctioned prior to administration of Exosurf Neonatal. Exosurf Neonatal suspension is administered via the sideport on the special endotracheal tube adapter **WITHOUT INTERRUPTING MECHANICAL VENTILATION.** Each Exosurf Neonatal dose is administered in two 2.5 mL/kg half-doses. Each half-dose is instilled slowly over 1 to 2 minutes (30 to 50 mechanical breaths) in small bursts timed with inspiration. After the first 2.5 mL/kg half-dose is administered in the midline position, the infant's head and torso are turned 45° to the **right** for 30 seconds while mechanical ventilation is continued. After the infant is returned to the midline position, the second 2.5 mL/kg half-dose is given in an identical fashion over another 1 to 2 minutes. The infant's head and torso are then turned 45° to the **left** for 30 seconds while mechanical ventilation is continued, and the infant is then turned back to the midline position. These maneuvers allow gravity to assist in the distribution of Exosurf Neonatal in the lungs. During dosing, heart rate, color, chest expansion, facial expressions, the oximeter, and the endotracheal tube patency and position should be monitored. **Suctioning should not be performed for two hours after Exosurf Neonatal is administered, except when dictated by clinical necessity.**

HOW SUPPLIED: Exosurf Neonatal for Intratracheal Suspension is supplied in a carton containing one 10 mL vial of Exosurf Neonatal for Intratracheal Suspension, one 10 mL vial of Sterile Water for Injection, and five endotracheal tube adapters (2.5, 3.0, 3.5, 4.0, and 4.5 mm I.D.). (NDC 0081-0207-01). Store Exosurf Neonatal for Intratracheal Suspension at 15° to 30°C (59° to 86°F) in a dry place.

EDUCATIONAL MATERIAL: A videotape on dosing is available from your Burroughs Wellcome Co. representative. This videotape demonstrates techniques for safe administration of Exosurf Neonatal and should be viewed by health care professionals who will administer the drug.

Licensed under U.S. Patent Nos. 4312860 and 4826821 500009

References: 1. Long W, Thompson T, Sundell H, et al. Effects of two rescue doses of a synthetic surfactant on mortality rate and survival without bronchopulmonary dysplasia in 700- to 1350-gram infants with respiratory distress syndrome. *J Pediatr* 1991;118:595-605. 2. Long W, Corbet A, Cotton R, et al. A controlled trial of synthetic surfactant in infants weighing 1250 g or more with respiratory distress syndrome. *N Engl J Med* 1991;325:1696-1703. 3. Speer CP, Robertson B, Curstedt T, et al. Randomized European multicenter trial of surfactant replacement therapy for severe neonatal respiratory distress syndrome: single versus multiple doses of Curosurf. *Pediatrics* 1992;89:13-20. 4. Horbar JD, Soll RF, Schachinger H, et al. A European multicenter randomized controlled trial of single dose surfactant therapy for idiopathic respiratory distress syndrome. *Eur J Pediatr* 1990;149:416-423. 5. Cowan F, Whitelaw A, Wertheim D, Silverman M. Cerebral blood flow velocity changes after rapid administration of surfactant. *Arch Dis Child* 1991;66:1105-1109. 6. Russell L, White A, Andrews E, et al. Observational study of synthetic surfactant in 11,455 infants. Presented at the 1992 Meeting of the American Pediatric Society/Society for Pediatric Research, May 4-7, 1992, Baltimore, MD. 7. Stevenson D, Walther F, Long W, et al. Controlled trial of a single dose of synthetic surfactant at birth in premature infants weighing 500 to 699 grams. *J Pediatr* 1992;120:S3-S12. 8. Corbet A, Bucciarelli R, Goldman S, et al. Decreased mortality rate among small premature infants treated at birth with a single dose of synthetic surfactant: a multicenter controlled trial. *J Pediatr* 1991;118:277-284.

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EX-YO3216

cluded methicillin-resistant *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and 10 genera of gram-negative bacilli. Colonization by respiratory pathogens was statistically associated with concomitant antibiotic therapy within the medical ICU group of patients, but not with severity of illness. Although medical ICU patients tended to have more dental plaque than preventive dentistry clinic patients, there was no statistically significant association noted between the presence of dental plaque and respiratory pathogen colonization. CONCLUSIONS: These findings suggest that bacteria commonly causing nosocomial pneumonia colonize the dental plaque and oral mucosa of intensive care patients. In many cases, this colonization occurs by large numbers of bacteria. Dental plaque may be an important reservoir of these pathogens in medical ICU patients. Efforts to improve oral hygiene in medical ICU patients could reduce plaque load and possibly reduce oropharyngeal colonization.

A Bench Test Evaluation of a Neonatal Closed Tracheal Suction System—FJ Monaco, KS Meredith. Pediatr Pulmonol 1992;13:121.

SUMMARY: We tested the hypothesis that a possible mechanism for reduced hypoxia during suctioning with closed tracheal suction systems (CTSS) is the provision of uninterrupted gas delivery and the maintenance of airway pressures. This was accomplished by using a neonatal ventilator and test lung model to permit the insertion of a CTSS (with and without suction applied), measurements of inspiratory and end-expiratory airway pressures proximal and distal to the endotracheal tube (ETT) and measurements of inspired and expired tidal volumes (sampled distal to the ETT). An 8-Fr CTSS was evaluated with 4.5, 4.0, and 3.5 mm ETT, and a 6-Fr CTSS with a 3.5, 3.0, and 2.5 mm ETT. We found that catheter placement without suction caused no changes in proximal airway pressures but distal peak inspiratory pressures decreased as the relationship between suction catheter size and ETT lumen increased, while distal end-expiratory pressures were unchanged. Similarly, distal tidal volumes progressively decreased as the size of the suction catheter occupied more of the ETT lumen. During suctioning with the 6-Fr catheter, proximal airway pressures were virtually unchanged; however, as ETT size decreased, distal airway pressures also decreased. The application of suction only modestly augmented the substantial decrement in measured tidal volumes when compared to catheter insertion alone. For the 8-Fr catheter, suction markedly reduced both proximal and distal airway pressures and tidal volumes. We conclude that the use of the CTSS tested in this neonatal ventilator/test lung model does not preserve continuity of volume or pressure delivery during suction procedures; therefore, these cannot be the sole explanation for the reported reduction in suction-related hypoxia.

Active Compression-Decompression: A New Method of Cardiopulmonary Resuscitation—TJ Cohen, KJ Tucker, KG Lurie, RF Redberg, JP Dutton, KA Dwyer, et al. JAMA 1992;267:2916.

OBJECTIVE: To describe and compare with standard cardiopulmonary resuscitation (CPR) in humans a new form of CPR that involves both active compression and active decompression of the chest. DESIGN: Patients in cardiac arrest in whom standard advanced cardiac life support failed were randomized to receive 2 minutes of either standard or active compression-decompression (ACD) CPR using a custom, hand-held suction device, followed by 2 minutes of the alternate technique. The ACD device was applied midsternum and used to perform CPR according to the guidelines of the American Heart Association: 80 compressions/minute, compression depth of 3.8-5 cm, 50% duty cycle, and constant-volume ventilation. Mechanical Thumper CPR was also compared in 5 patients. End-tidal carbon dioxide ($etCO_2$) concentrations and hemodynamic variables were measured. Transesophageal Doppler echocardiography was used to assess contractility, the velocity time integral (an analogue of cardiac output), and diastolic myocardial filling times. RESULTS: 10 patients were enrolled. The mean (SD) $etCO_2$ was 4.3 (3.8) torr with standard CPR and 9.0 (3.9) torr with ACD CPR ($p < 0.0001$). Systolic arterial pressure with standard CPR was 52.5 (14.0) torr and with ACD CPR, 88.9 (24.7) torr ($p < 0.003$). The velocity time integral increased from 7.3 (2.6) cm with standard CPR to 17.5 (5.6) cm with ACD CPR ($p < 0.0001$), and diastolic filling times increased from 0.23 (0.09) seconds with standard CPR to 0.37 (0.12) seconds with ACD CPR ($p < 0.004$). Mechanical Thumper CPR consistently underperformed both standard and ACD CPR. Minute ventilation obtained in 4 patients during ACD CPR without endotracheal ventilation was 6.6 (0.9) L/min. After 1 hour of standard CPR failed, 3 of 10 patients randomized to ACD CPR rapidly converted to a hemodynamically stable rhythm following 2 minutes of ACD CPR. CONCLUSION: ACD CPR is a simple manual technique that improved cardiopulmonary circulation in 10 patients during cardiac arrest. Although ACD CPR may have produced a return of spontaneous circulation in 3 patients refractory to standard measures, its impact on survival when used early in cardiac arrest remains to be determined.

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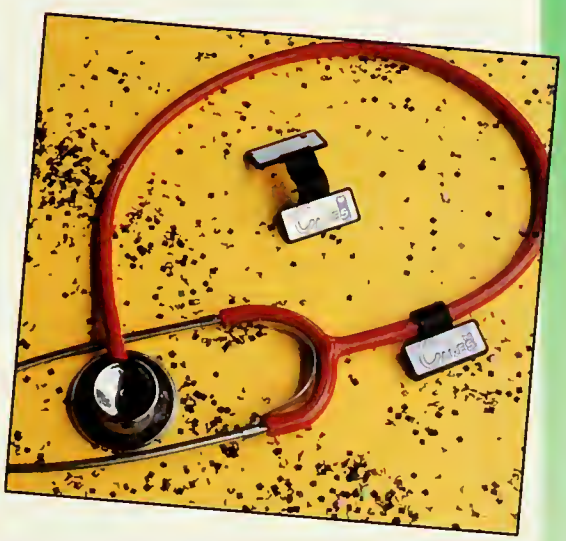

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$51 to $75	$5.50	$13.00	$31.00
$76 to $100	$7.00	$16.00	$38.00
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More Clinical Practice Guidelines: Now What?

In December 1991, the first five AARC Clinical Practice Guidelines (CPGs) were published after 2 years of work by the CPG Steering Committee and its Working Groups.[1-6] In this issue of RESPIRATORY CARE, the second group of CPGs is published. The same methodology was used for these CPGs as that used with the original group. CPGs are not published until they have been carefully scrutinized by the Working Groups, the CPG Steering Committee, and hundreds of persons who serve on the panel of reviewers. The first CPGs have been well received by the respiratory care profession.

Thanks for the Help

This project has been the result of the hard work of many persons, each of whom deserves the thanks of the entire respiratory care profession. The primary responsibility for development of CPGs rests with the Working Groups and their chairpersons. However, "rests" is a poor choice of word here, because the Working Groups have had little chance to rest. Their efforts are seen on the pages of this issue of the Journal. The five Working Groups and their chairpersons are Oxygen Therapy (Dianne Lewis MS RRT), Aerosol Therapy (Jon Nilsestuen PhD RRT), Mechanical Ventilation (Richard Branson RRT), Bronchial Hygiene (Lana Hilling CRTT), and Cardiopulmonary Diagnostics (Kevin Shrake MA RRT). The CPG Steering Committee is composed of these persons and Dean Hess MEd RRT (chairman), Jerome Sullivan MS RRT, Patrick Dunne MEd RRT, Sam Giordano MBA RRT, Pat Brougher RRT, Neil MacIntyre MD, and James Stoller MD.

The efforts of the Association members and friends of the respiratory care profession also deserve recognition and thanks. Almost 700 persons serve as volunteer reviewers of CPGs (obviously, this list is too long to publish). The support of the elected leadership of the AARC office and the Executive Staff is appreciated; without this support, a project of this magnitude would not be possible. The publication of 13 CPGs speaks well for the maturity of our profession. Indeed, this demonstrates professional leadership to our health care colleagues in our sister professions.

Now What?

We challenge the profession to incorporate the CPGs into institution-specific protocols and algorithms—to convert the relatively abstract guidelines to pragmatic, utilitarian applications with the potential to improve patient care and delineate and increase practitioner responsibility. Further, we suggest that an objective and systematic method of measuring the impact of such protocols be incorporated from the outset. This could be in the form of a quality improvement project using the JCAHO model, with the results shared with peers as an AARC OPEN FORUM abstract in 1993. If enough such abstracts are received and judged acceptable, a special symposium can be devoted to their presentation at the 1993 AARC Annual Meeting. We hope that many of you will accept this challenge. In the final analysis, the success of the AARC CPG project rests on such outcome-oriented reports of its successes (and failures). Although not specifically related to CPGs, an example of such a project published in this journal is described by Beasley et al[7] addressing blood gas utilization. The paper by Hadorn et al[8] published in JAMA also describes a potentially valuable approach.

More to Come

The AARC remains committed to this project, and CPGs will continue to be developed and published. What remains to be established is the impact of the CPGs on the practice of respiratory care. The nature and extent of that impact can be determined only by appropriately designed studies.

Dean Hess MEd RRT
Chairman
Clinical Practice Guidelines Committee

Assistant Director
Department of Research
York Hospital
York, Pennsylvania

Pat Brougher RRT
Editor
RESPIRATORY CARE
Clinical Practice Guidelines Project Manager
Dallas, Texas

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On Mythology and Naked Emperors: What Are You Doing With All Those Numbers You're Writing Down?

Over the years, respiratory care practitioners (RCPs) have learned 'facts' that they later had to unlearn after their validity had been challenged by objective scientific inquiry. As Shakespeare commented,

> Yea, from the table of my memory,
> I'll wipe away all trivial fond records.[1]

Remember this hallowed bit of gospel? "IPPB pushes the medication in deeper." Yeah, right. And this one: "Never turn the pressure higher than 15 or 20 cm H_2O for an IPPB treatment." (When I would hear such an answer during the Registry oral exams, I would then ask the therapist/candidate, "And so, if this same patient went bad and had to be intubated and put on a ventilator, you'd set the pressure at 15?" Amazing, the back-pedaling answers we'd get.)

Here's a classic: "IMV makes the patient work harder than if he were on assist/control." Not unless you're using a lousy IMV setup, says Dr John Downs, one of the 'inventors' of IMV.[2] Dr Downs has also said that if your patient is working harder to breathe on your IMV system than he was when not attached to it, then please call your invention something other than IMV, thank you very much (JB Downs, personal communication. Guest professor's ICU rounds, The Johns Hopkins Hospital, Baltimore MD, 1978). Real IMV—with or without that overglorified S, as in SIMV[3,4]—doesn't make the patient suck back through 3 feet of tubing to open a one-way valve; nor should demand-valve IMV, built into a ventilator, cause a patient to work hard and/or wait during a sluggish response time for that gas to be delivered.[2]

How about this one: "PEEP always decreases venous return and makes the blood pressure drop." Hmmm. I wonder, then, why those patients on properly used PEEP get better instead of worse.

Here's a related one: "PEEP above 10 or 15 cm H_2O is very dangerous. Never go above 15." Double Hmmm. I wonder why virtually all modern ventilators go up to 40 cm H_2O of PEEP (or more) as standard equipment. *Somebody* is using pressures that high, and with great success, and it's not new.[5] There must be a lot of clinicians doing it, or such PEEP pressures wouldn't be built into ventilators as a standard feature. (I personally have used 50 cm H_2O of PEEP on a couple of ARDS patients. The results were dramatic and wonderful. The alternative, each time, would have been to stand by with wringing hands and watch death from hypoxemia.)

My point is that many of us have been the victims of pseudoscientific hogwash over the years, and much of it has been our own fault. Too often we have been reduced to being obedient robots, never challenging the erroneous pontifications that came from *Them*, those mysterious Experts who offered so much of that early inhalation therapy wisdom that was based on guess, conjecture, erroneous methods, inadequate equipment, and other nonscience. Example: In the early days of PEEP, if 'high' levels of positive end-expiratory pressure were attempted on patients on assist/control, then *of course* they blew a lot of lungs and saw a lot of hypotension, especially if the ventilator was an MA-1, a notoriously weak machine that takes until next Tuesday to deliver the breath and get it over with.[6-8] But isn't it interesting that during that same pioneering time, researchers using more powerful ventilators, including those venerable old Emersons and Engstroms and the early Servos, had a great deal of success with what was once called super-PEEP? The point is, PEEP wasn't at fault for those early

disasters (and, alas, some modern-day ones); its improper application was. It's rather like saying that no one should receive anesthesia for surgery because lots of patients died during the early days of anesthesia. Yet that kind of illogic is exactly what we sometimes find ourselves working against when we try to do our best for patients.

On the other hand, much of what we were taught early in our professional educations is classic, quintessential stuff that should be forever useful and edifying. A major task for all of us, therefore, would be to separate the hearsay from the proven, the science from the nonscience.

Possibly the most egregious example of that kind of nonscience is the reliance on, and fear of, peak inspiratory pressures when a patient is on a ventilator. We learn as students that *plateau* pressure is pretty close to alveolar pressure because it's a static measurement—a reading taken during a time of no flow, when pressures are equalized between alveoli and manometer.[9] That's why compliance is calculated by dividing the tidal volume by the plateau pressure. That is the pressure that was required to move that much air into the lungs.

We also learn as students that the *peak* inspiratory pressure is the sum of the alveolar (plateau) pressure and the driving pressure of the ventilator to move that gas through the tubing system, through the endotracheal tube (ETT), and through the patient's airways.[10] Peak pressure, therefore, is dynamic, as opposed to static, and as such cannot be used to measure compliance because by definition compliance is a static measurement. Example: Let's say that the set tidal volume is 800 mL, and we find that the plateau pressure is 40 cm H_2O, and the peak pressure is 60 cm H_2O. 800/40 = 20 cm H_2O pressure to move each mL of air into the patient's lungs. The peak pressure of 60 tells us that our patient required 20 cm H_2O more (60 – 40) pressure to overcome the resistance of the ventilator tubing, the ETT, and the bronchi.

Now, let's suppose that the ETT kinks, or that the patient needs suctioning, or that some other event occurs that has nothing to do with alveolar compliance—and that now we see that the peak pressure has increased to 70 cm H_2O. When we measure the plateau pressure, however, we see that it's still 40 cm H_2O. In other words, the patient's compliance is exactly the same, 800/40. If we were to divide the tidal volume by the peak pressures (originally 60 and now 70), we would erroneously report that the patient's compliance had worsened.

Peak inspiratory pressure gives us useful information about resistance, not compliance.[11] In the above example, the higher pressures would alert us to the kinked ETT. Suppose, however, that nothing is wrong with the ETT, and the patient is not in bronchospasm, and he doesn't need suctioning. But let's say that I don't like the patient's inspiratory-expiratory ratio (I:E), and we've determined that this patient does best with a nice long expiratory time, with an I:E of 1:4 or 1:5—with that long expiratory phase allowing minimal interference with venous return and blood pressure—and with lower mean airway pressure (more on that later) and less chance of pneumothorax and other barotrauma. So I increase the peak flow on the ventilator and achieve the desired shortened "I" and lengthened "E," and the peak pressure coincidentally increases from 60 to 70. Well, some folks will throw up their hands and exclaim, "Oh, my goodness! The peak pressure has gone up!" My response is, "So what? Who cares?"

Those of us who give such an answer are sometimes looked at askance, as if we're being flip or incautious or unfeeling. *Au contraire.* Of course the peak pressure increased when the peak flow was turned up; the ventilator is now blowing harder to push the same tidal volume through its tubing system, and that's what needs to be remembered. Within realistic limits, most of that increase in peak pressure caused by a careful increase in peak flow is occurring in the ventilator tubing. Some really bad lungs are so noncompliant that powerful peak flows, up to 150 L/min or more, have been recommended in order to deliver the inspiration in enough time to maintain an adequate I-E ratio.[12] Such a fast flow will perforce raise the pressure in the tubing system, but it is a pressure rise that can be safely controlled and monitored by the careful clinician. In our hypothetical example, for instance, our patient's plateau pressure—his lung pressure—might remain at 40 cm H_2O. Also, some of the newer ventilators give you a handy readout of mean airway pressure, a measurement that used to be cumbersome and tedious and nearly impossible to make.

I predict that when the peak flow is increased to provide a good, adequate I:E, you will see both the plateau pressure and the mean airway pressure *stay the same or even decrease (improve), because there is now a longer expiratory time during which lung pressures are allowed to fall toward baseline.* And, yes, the peak pressure went up. I repeat, So what? Who cares if the pressure in the plastic tubing is higher, if you know what you're doing? Yes, I'm excluding compressible volume (tubing compliance) in this example, but even if you calculate that volume difference before and after increasing the peak flow, you will still see that *lung* compliance has either stayed the same or even improved slightly. To quote Pierson, "Static inflation-hold pressure more accurately reflects alveolar conditions than does PAP [peak airway pressure], and should be used as the primary monitor of the risk for alveolar rupture."[13] Yet I can't even count how often I've seen the perverted, bizarre situation wherein a patient is given an inadequate flow resulting in an I:E in the 1:1 range, because a faster peak flow would have made the peak pressure rise (so what?), and then everybody wonders why the patient is doing so poorly. Hey! give the poor guy some decent expiratory time and watch the difference!

Now don't misunderstand, and please don't misquote me. I did not say that peak pressure is useless or innocuous. In fact, the above examples clearly spell out the usefulness of peak pressures, and caution must be used whenever any ventilatory change is effected. Even some who advocate a timid approach to increasing peak flows because of a fear of peak pressure-related barotrauma, however, do not support their concerns with data that include the I:E in use during lung injuries.[14] To blame an increase in peak pressure alone for a pneumothorax or some other lung injury is irresponsible. Was there a long enough *machine* expiratory time so that the *lung* I:E was adequate? If the ventilator in use was not one that automatically adjusts peak flow to maintain I:E when other changes are made, was the peak flow manually adjusted properly? Was the patient on assist/control? Was there a concomitant decrease in mechanical tidal volume if high levels of PEEP were necessary? Is the patient in status asthmaticus? Any of these variables must be addressed when one is dealing with peak flows and peak pressures, and I am in no way advocating the indiscriminate jerking around of *any* ventilator setting or resultant measurement. If that's not clear, then please re-read all of the above, starting with the first paragraph.

(And, speaking of ventilator settings, can anyone out there tell me exactly when the mathematical and statistical term "parameter" began its widespread incorrect usage, particularly in respiratory therapy? I believe it was right around the same time that "hopefully" began its dreadful misuse, along with "between you and I," which was about the same time as "healthy" began to be used for "healthful." If you know, please write.)

I suspect that the history of this unwarranted and illogical concern over peak pressures can be traced to three things: the term "dynamic compliance," which is an oxymoron; the mythology surrounding laminar flow; and some incomplete understandings of inspiratory flow curves, which on modern ventilators are easy to play with.

As already mentioned, compliance is by definition a static measurement, and peak pressure is a nonstatic event. Therefore, dividing tidal volume by peak pressure gives a rather useless number. Peak pressure is useful and important in calculating resistance, not compliance, but how many of us actually do that (although we should)? And just as importantly, if we do have a ventilator with all the bells and whistles required to measure resistance, what do we do with that potentially very valuable information? Something, I hope. I've see an awful lot of dutiful number-scribbling done on ventilator flowsheets, with all these neat measurements being recorded and then ignored.

And then there's laminar flow. I wonder how many RCPs reading this were once taught that peak flows $\leq$ 40 L/min are laminar, and flows > 40 L/min are turbulent? Absolutely untrue, and later I'll address where that myth probably originated. For now, let's again return to our student days. Remember such delightful stuff as Reynolds' number? The Hagen-Poiseuille law? The viscosity of the gas? The density of the gas? The diameter of the tube(s) the gas is flowing through? The liter flow of the gas? The critical velocity of the gas? *All* of those factors must be used in calculating laminar versus turbulent flow,[15] and I challenge anyone in

the world to look at some number on a ventilator peak-flow control and declare that *that* flow delivering *that* tidal volume with *that* oxygen-nitrogen mixture featuring *that* gas density and *that* gas viscosity going through *that* ETT and *those* bronchi at *that* moment is going to produce a Reynolds' number above or below 2,000. I would certainly like to shake the hand of such a seer, such a mystic, such a guru, such a prophet. No doubt that person could also tell us what Elvis is doing these days.

But the good news is that, below the lobar bronchi, almost all flow is laminar anyway.[16,17] To quote one source: In very small tubes of any length, the velocity of the gas cannot exceed the critical velocity at any pressure differential, and thus turbulent flow is impossible (eg, in the small bronchi and bronchioles of the lung).[18]

So, if anyone's really hung up on laminar flow, there it is. And, yes, at certain peak flow settings, the flow in the corrugated tubing might be turbulent, but my question, as you can guess by now, is, So what?

By the way, I strongly suspect that that magic number, 40 L/min, originated with a user's manual that came with a once-popular ventilator.[19] The manual *suggested* that peak flow be set at 40 L/min "to start." Unfortunately, the number 40 then became, to some clinicians, graven on stone tablets.

Another number that is measured, recorded, and then unfortunately ignored is oxygen content. We learn as students that most of the oxygen is carried on the hemoglobin; then we turn around and use P_{aO_2} as the major indicator of oxygenation. Ironically, most modern blood gas analyzers calculate the oxygen content for us, so we don't even have to do the arithmetic anymore: $1.34 \times Hb$, etc. Yet how often have you seen anemic, hypoxemic patients with great P_{aO_2}s whose hypoxia is ignored "because the P_{O_2} is good"?

Inspiratory flow curves are another example. Modern ventilators let us experiment with various flow patterns, but how many of us are using such information to belabor the obvious: that flow generators (square wave and true semi-sine wave) get the breath in and get it over with in the shortest possible inspiratory time, and pressure generators (ramp, or descending square wave, ascending flow) take longer for "I" at any given peak flow setting and therefore intrude on "E" time and on mean airway pressure? Oh, but I forgot: a square wave will raise the peak airway pressure. Did I mention, So what?

My point is simply this: As students we were taught that certain concepts were important, but once upon a time the practical use of those concepts seemed to be confined to large research centers with the technology and personnel to perform those seemingly arcane calculations. Now, many (most?) hospitals have ventilators and blood gas analyzers that spit out those numbers at the push of a button. We're paying for that technology, but are enough of us using it? Or is that too much trouble? I wonder whether some of us are paraphrasing what the Emperor said to Mozart about the number of notes in his music: "Well, there it is. Too many numbers."

A few years ago I attended a seminar on ventilators where an enthusiastic salesman was presenting his machine, full of microprocessed doodads and impressive computer readouts. His ventilator (which, by the way, does nothing that the Siemens Servo hasn't been doing for the past 20 years; he wasn't pleased when I pointed that out) is supposedly superior because you push buttons instead of turning knobs. Wow. When I repeatedly asked him what kind of *ventilator* it was, he either wouldn't or couldn't answer the question. I want to know how powerful a respirator is. How well does it push air into severely stiff or tight lungs? Remember that? Every time I'd ask him the question, he'd point to a computer screen or a key pad or a really swell alarm. "Behead the damn machine," I said. "Remove the bells and whistles and let's see what kind of blower is left to get the air into the lungs." The salesman still didn't get it, so I exasperatedly used an analogy: "Look, I keep asking you what kind of traction, limited-slip differentials, and climbing power this four-wheel-drive has, and you keep pointing to the nice upholstery and cute stereo."

He still didn't get it. And when I asked him what he advised therapists to do with all the neat numbers his machine was spitting out, he still didn't get it.

The animated cartoon character Homer Simpson works in a nuclear power plant. Part of his job is to take a clipboard with a flowsheet and record the

readings from the gauges on the nuclear reactor, perhaps q2h or so. A running gag on the series shows Homer busily writing down the numbers on his flowsheet, including when a manometer needle is in the red "danger" zone, but not bringing such readings to anyone's attention. After all, it's time for his donut break, or maybe it's almost time for his shift to be over. Alas, Homer will never realize which of those numbers are just busy-work writing exercises, and which are potentially useful and perhaps even lifesaving. How sad that he can't tell the difference. If he could, it might possibly make his job actually interesting and important.

Sound like anyone you know?

Tom Burns RRT
Instructor in Respiratory Care
GateWay Community College
Phoenix, Arizona

Reprints: Tom Burns RRT, GateWay Community College, Respiratory Care Dept, 108 N 40th St, Phoenix AZ 85034.

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An Evaluation of MDI Spacers and Adapters: Their Effect on the Respirable Volume of Medication

Jerry Ebert RRT, Alexander B Adams MPH RRT, and Beth Green-Eide MA RRT

The use of a metered dose inhaler (MDI) with an adapter or spacer designed to deliver medication to ventilated patients has been found to produce effects similar to those achieved with small volume nebulizers. For 9 commonly used MDI adapters or spacers, we measured and compared their production of available respirable volume (ARV) of medication. A simulated ventilator circuit was configured to determine ARV as measured by a laser particle-size counter. The adapters or spacers were grouped in three categories: chamber, in-line, and elbow styles. The devices were actuated into the circuit, and ARV was measured 36 separate times with various combinations of medication and medication canister. We found significant differences in ARV between devices, device categories, and medications as compared by ANOVA testing. In this study, chamber-style MDI devices produced greater respirable volume than the in-line- or the elbow-style devices. Of the chamber-style devices, the cone-shaped chamber device provided more respirable volume than did the other two chamber devices. We also found differences among the medications, although we did not anticipate such differences. The possible factors to explain medication differences were not investigated in this study. Because ARV differs according to the design of the individual device used, the choice of device may alter the respirable volume of the medication delivered and, therefore, the effectiveness of the prescribed medication. We conclude that to achieve the desired effects of the medication, practitioners may need to customize the number of MDI actuations according to the device in use. (Respir Care 1992;37:862-868.)

Introduction

Critically ill patients receiving mechanical ventilation are frequently prescribed aerosolized medications. Small volume nebulizers (SVN) have been the usual aerosolizing device, but recent reports suggest that the effect of aerosol administration by metered dose inhaler (MDI) is similar to that of the SVN.[1-8] More particularly, measurements of isorecoil flow (a measure of expiratory resistance) on ventilated patients have found similar improvements with the use of either device.[3] As MDI use in ventilated patients has increased, the refinement of techniques, delivery methods, and equipment adapters has been investigated. Also, recently, two studies have reported the use of intratracheal catheters to improve the delivery of medication from MDIs to intubated patients.[9,10] Crogan and Bishop have reported significantly more aerosol delivered when an MDI is activated at the beginning of the inspiratory phase of the ventilator cycle rather than before gas flow is initiated.[8] It is known that the endotracheal tube acts as an impediment to aerosol delivery by an MDI,[11] yet there are few studies comparing the efficacy of aerosol delivery beyond the endotracheal tube by various methods or adapters to administer MDIs. In general, specific performance characteristics of MDI adapters and their use have not been thoroughly evaluated.

The purpose of this study was to measure an efficacy characteristic of 9 commercially available

The authors are associated with St Paul-Ramsey Medical Center, Respiratory Care Services, St Paul, Minnesota.

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Reprints: Beth Green-Eide, Director of Respiratory Care Services, St Paul-Ramsey Medical Center, 640 Jackson St, St Paul MN 55101.

MDI adapters and spacers (Table 1).* We will term this characteristic the available respirable volume (ARV), the volume of medication delivered to a simulated circuit within the particle-size range usually deposited in the respiratory tract (0.67-5 μ) as measured by a laser particle-size counter. We compared the ARV delivered by four MDI medication canisters (Table 1).

Table 1. Metered Dose Inhaler (MDI) Adapters, Spacers, and Medications Evaluated for Available Respirable Volume

MDI Adapters and Spacers
ACE (Aerosol Cloud Enhancer)
Aerochamber
Aerovent
Hudson RCI
Instrumentation Industries
Baxter Airlife dispenser-adapter
Marquest ventilator elbow
Medicomp ventilator elbow
DHD ventilator elbow
Medications
Azmacort (triamcinolone acetonide)
Ventolin (albuterol)
Maxair (pirbuterol acetate)
Atrovent (ipratropium bromide)

Materials and Methods

We evaluated the 9 MDI adapters and spacers displayed in Figures 1, 2, and 3: the chamber-style spacer and the in-line- and elbow-style adapters, respectively. We compared the performance of these MDI adapters and spacers using a laser particle-size counter. MDI performance was based on the measurement of available respirable volume (ARV) defined as the volume delivered by particles in the respirable range of 0.67-5.0 μ assuming that the particles were spherical and their particle volume was calculated by multiplying the volume for that size sphere by the number of particles in that range. This calculation is described by Bishop et al.[12]

*Suppliers are identified in the Product Sources section at the end of the text.

Fig. 1. Chamber-style adapter and spacer devices listed from left to right: ACE (Aerosol Cloud Enhancer), Aerochamber, and Aerovent.

The airborne particle counter contains a laser diode aerosol sensor integrated with a digital in-line mass flowmeter and an 8-channel particle counter. The counter has an internal pump that draws air at 1 cfm (cubic feet/min). The circuit was configured as in Figure 4. The cuff of the endo-

Fig. 2. In-line MDI adapters listed from left to right: Hudson RCI MDI adapter, Instrumentation Industries MDI adapter, and Baxter Airlife dispenser-adapter.

Fig. 3. Elbow MDI adapters listed from left to right: Marquest ventilator elbow, Medicomp, and DHD ventilator elbow.

tracheal tube was inflated with 1.5 mL of air to confirm that air would not be entrained into the system. We interposed the 9 adapters and spacers, according to manufacturers' recommendations, prior to the patient Y or at the endotracheal tube adapter and the circuit, using appropriate connectors.

Each of the 9 MDI devices was tested 36 times. Each device was tested 3 times using 3 representative units (ie, 3 Aerochambers, 3 DHD). This procedure was repeated for the 4 different medications (Table 1). Each medication was evaluated by testing 3 canisters of each medication (ie, 3 canisters of albuterol). Multiple tests were made to assure that all devices of the same manufacturer functioned the same and that all medication canisters had a similar output. The procedure for testing was as follows: (1) The circuit was set up with a test device and thoroughly flushed with air washes to eliminate any latent particles in the circuit; (2) a canister of medication was vigorously shaken, placed in the adapter or spacer, and actuated; (3) the particle counter was turned on within 1 second of actuating the canister; (4) measurements were then made of particles in the respirable range; (5) Steps 1 through 4 were repeated with each of the remaining canisters of medication; (6) we repeated this process for the remaining two devices from the same manufacturer; and (7) the entire procedure was repeated for each MDI device with each of the 4 medications.

The results were analyzed by analysis of variance (ANOVA) using the MDI adapter/device and medication as factors. Significance was tested by a Scheffe's post hoc test to correct for multiple comparisons.[13]

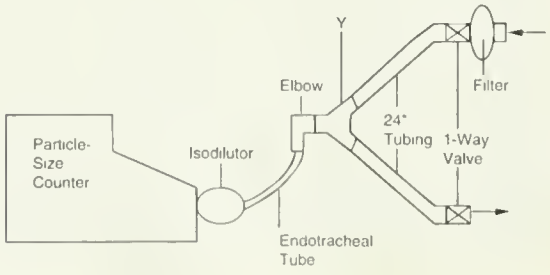

Fig. 4 Circuit for evaluating respirable volume of medication via MDI adapters and spacers.

Results

The MDI actuation delivery sessions appeared similar between medications and within sessions of device categories. However, medication impaction on the endotracheal tube was observed during use of the elbow devices. Impaction on the Y piece was noted during use of the in-line devices, while no impaction was observed during use of the chamber devices. Figure 5 represents the ARV delivered by each MDI device using the medication albuterol.

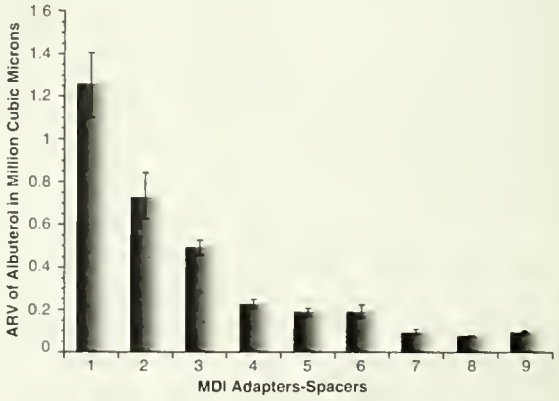

Fig. 5. Mean available respirable volumes (with standard deviation bars) of albuterol measured after actuation of the 9 MDI adapters and spacers tested. 1 = ACE; 2 = Aerochamber; 3 = Aerovent; 4 = Hudson RCI; 5 = Instrumentation Industries; 6 = Baxter Airlife dispenser; 7 = Marquest ventilator elbow; 8 = Medicomp ventilator elbow; and 9 = DHD ventilator elbow.

Figure 6 shows that the style of device produces a different mean ARV; the three chamber or spacer-style MDI adapters deliver a greater average amount of ARV than either the in-line ($p < 0.001$) or the elbow-type adapters ($p < 0.001$). Of the chamber-spacer style group devices, Device 1 delivered more ARV of albuterol than Device 2 ($p < 0.01$) or Device 3 ($p < 0.001$). Figure 7 dis-

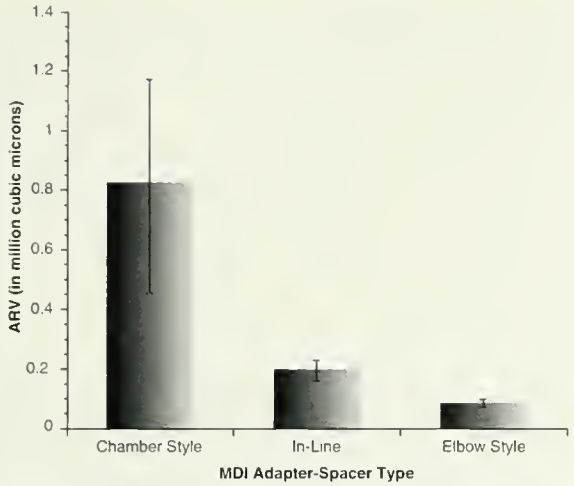

Fig. 6. Mean available respirable volumes (with standard deviation bars) measured after use of the three styles of MDI spacers and adapters tested.

plays the performance of the 9 adapters for the other 3 medications evaluated by displaying column comparisons and mean ARV values. Whereas the differences in ARV production between canisters was small (note the standard deviations in Figures 5 and 6), a similar ARV production profile to the albuterol pattern can be seen for the other medications (Fig. 7).

When analyzed across all devices, a statistically significant difference was found only between Maxair and Azmacort (Maxair having a greater ARV than Azmacort, $p < 0.001$). Table 2 presents the two-way ANOVA results testing the main effects of the MDI adapters (devices) and medications. The greatest effect was due to significant differences between devices, but medication differences were also significant. A small but statistically significant interaction was found between device type and medication. This interaction became insignificant if Azmacort was dropped from the analysis. Therefore, the uniformly lower ARV measured from Azmacort is primarily responsible for the interaction.

Discussion

Recently, it has been shown that the use of aerosolized medication delivery by an MDI can have nearly equivalent effects to SVN. Therefore, interest has increased in the cost saving, convenience, and performance of MDI adapters. Hess has reviewed the operation and cost of commercially available MDI adapters for ventilator circuits,[14] summarizing the methods and types of adapters in current use and making recommendations for the appropriate use of MDI administration in mechanically ventilated patients. Bishop et al[12] compared the respirable volume of aerosol delivered by 3 commercially available adapters as measured by a

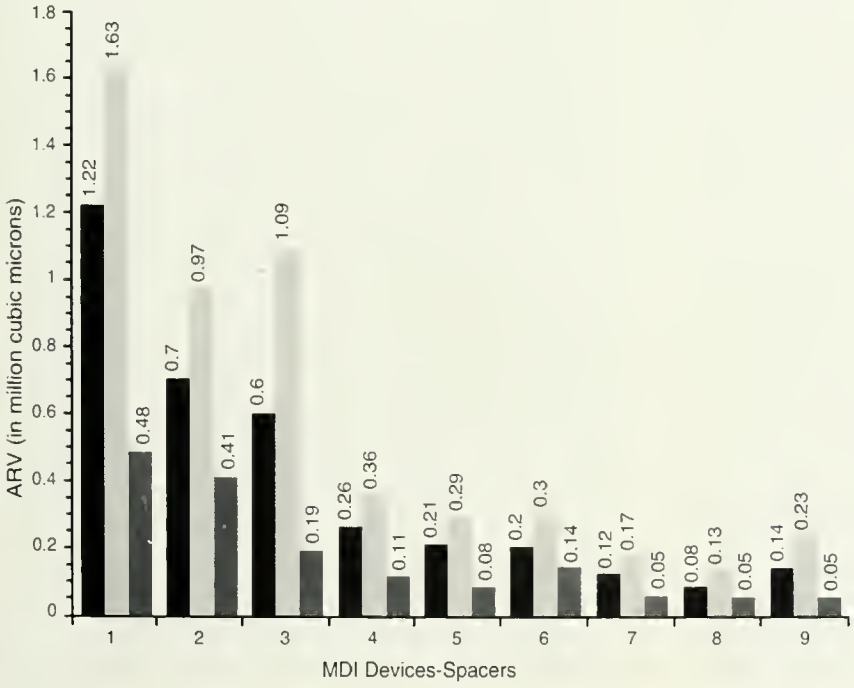

Fig. 7. Available respirable volume measured after actuating medication canisters of Atrovent ■, Maxair ■, and Azmacort ■ through the 9 MDI adapters and spacers tested. 1 = ACE; 2 = Aerochamber; 3 = Aerovent; 4 = Hudson RCI; 5 = Instrumentation Industries; 6 = Baxter Airlife dispenser; 7 = Marquest ventilator elbow; 8 = Medicomp ventilator elbow; and 9 = DHD ventilator elbow.

Table 2. Analysis of Variance Table of the Main Effects of Devices, Medications, and Interactions upon Available Respirable Volume

Source	Degrees of Freedom	Sum of Squares	Mean Square	F Value	p Value
Medications (A)	3	2.174	0.725	205.327	< 0.0001
Devices (B)	8	12.582	1.573	445.634	< 0.0001
Interactions (AB)	24	1.936	0.081	22.857	< 0.0001
Residual	72	0.254	0.004		

laser particle-size counter. They reported that the aerosol delivered using MDI adapters varied with the adapter and recommended that the number of actuations be adjusted according to the adapter in use. In general, their suggestion was to increase the number of actuations per medication administration session. This bench study further investigated the performance of MDI adapters by measuring the ARV for 9 devices when 4 different medications are used.

This study attempted to standardize several aspects of an MDI delivery system by using a fixed, simulated ventilation circuit and endotracheal tube system. When the 9 MDI adapters and spacers were evaluated for their ARV, differences in respirable volume were measured. The devices were grouped in categories of style and shape; differences were found between the groups in their production of respirable volume of medication. More ARV was made available by the chamber-device group than the in-line and elbow-style devices. It was visually observed that there was significant impaction of the aerosolized medication on the patient Y with the in-line device and in the endotracheal tube with the elbow device. This was not observed after MDI actuation into the chamber-style devices, and this difference could partially account for the greater ARV measured from the chamber-style devices. Also, the larger chamber volume of the chamber-style devices provides an increased space for accumulation of aerosolized medication particles and a decreased probability for impaction. Of the three chamber devices, Device 1 had a greater ARV than the other two chamber-style devices. This may be due to two factors: (1) the design of Device 1 directs the aerosol retrograde in the circuit, possibly avoiding immediate impaction and rainout, and (2) the shape of Device 1 is conical, not tubular, possibly reducing areas of sequestered aerosol. Also, Device 2 had a greater ARV than Device 3. The difference in ARV between Devices 2 and 3 may be due to the design of Device 3, which allows particles to collect in areas outside the main flow of gas.

Although not expected, differences were found in ARV between medications. Averaging over all the MDI devices tested, Maxair had a greater ARV than Azmacort ($p < 0.003$). When the variation due to MDI device was excluded, Maxair had a mean ARV greater than the other 3 medications and Azmacort had a mean ARV less than the other 3 medications. Because the study was not originally designed to evaluate differences between medications, we can only speculate on possible causes for the differences. ARV differences between medications could be due to differences in canister size, canister pressure, medication concentration or molecular characteristics of the medication, nozzle style or composition (metal vs plastic), or actuation technique. These possibilities could partially account for MDI device differences, but the variability due to medication differences was much less than that due to the MDI devices.

Realizing that this was a bench study of one aspect of MDI adapter performance, analogies made to clinical performance of the MDI adapters and spacers may not be valid. Compared to performance in the clinical setting, the conditions in which the devices were evaluated were relatively static (ie, the effects of humidification, ventilator volume, flowrate, frequency, or pause time on ARV were not evaluated). Therefore, how these devices may perform with a ventilator, with different ventilators, with different ventilator circuits, or at various ventilator settings was not measured and cannot be fairly speculated upon. In addition, other comparisons to their possible performance in the clinical setting were not evaluated (such as the ef-

fects on ARV upon encountering circuit or airway bifurcations, airway resistance sites, or mean or peak pressure changes). Because this evaluation was a laboratory study, we did not measure important clinical outcome variables used to assess aerosolized medication effectiveness such as isorecoil flow, peak flow, changes in airway resistance, breath sounds, or peak and mean airway pressures. We did not compare expense, convenience, potential dead space, or compressible volume factors of the devices. Another limitation is that the laser particle-size counter aspirates gas, whereas a cycling ventilator forces gas into the patient. Finally, because the counter has been used in only one previous study,[12] problems with its technique or accuracy may not have been thoroughly examined.

Nevertheless, as a measurement of one aspect of medication delivery by MDI, we carefully measured and compared the availability of aerosolized particles in the respirable range for 9 MDI adapters and spacers. We found significant differences in particle availability between devices and device groups as compared by analysis of variance (ANOVA). In this study, chamber- or spacer-style MDI devices produced greater respirable volume. In agreement with the Bishop study,[12] because ARV differs among devices used, the choice of device may alter the volume delivery and effectiveness of the prescribed medication. If this performance characteristic is known to be less for the device in use, more actuations may be necessary for a desired effect. If more respirable volume is known to be delivered by the device in use, there is a greater opportunity for the medication to be effective, and fewer MDI actuations may be necessary for a desired effect. To extend the observations of this study to the clinical setting, studies of physiologic response to medication delivery through various MDI adapters should be conducted in a clinical setting. Because physiologic response can be difficult to measure on ventilated patients, this bench study was undertaken to safely measure a characteristic of MDI adapters prior to clinical studies.

ACKNOWLEDGMENTS

We thank Jon Langenfeld for his help in gathering information for this paper.

PRODUCT SOURCES

Laser Particle-Size Counter:

Hiac/Royco 5250, Hiac/Royco, Silver Spring MD

Connectors:

Silicon adapter, Marquest # 17200, Marquest Medical, Englewood CO

22-mm adapter, Marquest # 177075, Marquest Medical, Englewood CO

Adapters and Spacers:

ACE (Aerosol Cloud Enhancer), DHD #11-1010, DHD Medical, Canastota NY

Aerochamber, Monaghan #82550, Monaghan Medical, Plattsburgh NY

Aerovent, Monaghan Medical, Plattsburgh NY

Hudson RCI MDI adapter, Hudson RCE #1659, Hudson RCI, Temecula CA

Instrumentation Industries MDI adapter, iii #RTC23-A, Instrumentation Industries, Bethel Park PA

Baxter Airlife dispenser-adapter, Baxter #002063, Baxter Healthcare Corp (Pharmaseal Div), Valencia CA

Marquest ventilator elbow, Marquest #178710, Marquest Medical, Englewood CO

Medicomp ventilator elbow, Medicomp #MD-1423, Medicomp, Coon Rapids MN

DHD ventilator elbow, DHD #60-0209, DHD Medical, Canastota NY

Medications:

Azmacort (triamcinolone acetonide), Rhone-Poulenc Rorer Pharmaceuticals, Fort Washington PA

Ventolin (albuterol), Allen & Hanburys, Research Triangle Park NC

Maxair (pirbuterol acetate), 3M Riker, Northridge CA

Atrovent (ipratropium bromide), Boehringer Ingelheim Pharmaceuticals, Ridgefield CT

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Evaluation of the Ability of the Syncoxy Breath-Synchronized Valve To Provide Adequate Oxygen Levels

Toru Sato MD DMSci, Naoto Okazaki DMSci, Noriki Saito MD DMSci, and Toshihisa Hasegawa

BACKGROUND: In recent years, interest has been focused on devices designed to minimize the volume of oxygen wasted during administration in order to decrease costs and increase the time that the ambulatory patient can be away from his major oxygen source. MATERIALS & METHOD: We compared the effects on oxygenation of breathing oxygen (1) supplied at 4 flowrates from a continuous-flow device (CLD) and from a Syncoxy breath-synchronized valve (SBSV) equipped with 3 different reservoirs in 12 healthy volunteers and in 24 patients with chronic obstructive pulmonary disease; (2) from a CLD, an Oxymizer Pendant, an Oxymizer Mustache, and a SBSV, at various flows, in 12 healthy volunteers; (3) from the breath-synchronized Oxymatic and a SBSV in 8 healthy volunteers; and we measured (4) the oxygen concentrations obtained at various outflows with the SBSV attached to a 2 L/min pressure-swing-adsorption oxygen concentrator. RESULTS: When compared to a conventional continuous-flow cannula, the SBSV afforded higher levels of oxygenation at equivalent flowrates or equivalent levels of oxygenation at flowrates reduced 30% or more. In addition, higher transcutaneous oxygen partial pressures were seen when the effect of SBSV on oxygenation was compared to the effects of other reservoir devices at the same flowrate. The addition of the SBSV to the concentrator allowed delivery of higher oxygen concentrations at higher flowrates. CONCLUSION: The SBSV can provide equivalent levels of oxygenation with less total oxygen usage—thus allowing greater patient mobility. (Respir Care 1992;37:869-876.)

Introduction

A number of studies[1-3] have established the value of supplemental oxygen administration in the management of chronic obstructive pulmonary disease (COPD). Continuous-flow oxygen through a nasal cannula is commonly used for this purpose in daily practice because it is a simple, comfortable, and effective method of oxygen administration. However, with continuous delivery, a considerable amount of the delivered oxygen (as much as 50-75%) may be wasted during expiration—increasing the cost and limiting the time that the ambulatory patient can be away from a large-capacity source of gas. In recent years, interest has focused on the development and evaluation of devices that conserve the oxygen supply by delivering oxygen only when triggered by an inspiratory effort and by storing the gas that flows during expiration for later utilization.[4-9]

Description of Device

We evaluated a small electronically controlled breath-synchronized valve called the Syncoxy* de-

Dr Sato is Professor and Chairman, Dr Okazaki and Dr Saito are Lecturers, and Mr Hasegawa is a technician in the Department of Anesthesiology, Tottori University Hospital, Yonago, Japan.

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Reprints: Toru Sato MD, Dept of Anesthesiology, Tottori University Hospital, 36-1 Nishi-machi, Yonago 683, Japan.

* Suppliers are identified in the Product Sources section at the end of the text.

signed to allow intermittent oxygen delivery and to deliver its peak flow during the initial phase of each inspiration (Fig. 1).[10] The Syncoxy (SBSV) consists of an electronic controller that drives a solenoid valve in the oxygen pathway, a respiratory sensor (or two sensors in series, in the production model) installed at the tip of a nasal cannula, and a reservoir tank. The device is powered either by batteries (4 AA dry batteries, for 130 hours of operation) or by 120-volt alternating current (ie, 'mains' line, or 'house' current). Any oxygen source can be used—oxygen cylinders, piped oxygen, or oxygen concentrator.

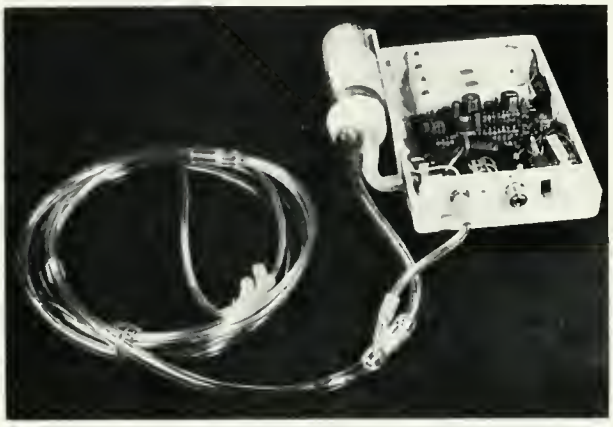

Fig. 1. The Syncoxy equipped with a small humidifier containing a sponge and a nasal cannula with sensor installed, registered in Japan as an approved medical device (No. 01B 1412, September 6, 1989). The case (which measures 92 x 155 x 33 mm) is shown open. From right to left on the front panel can be seen (1) a small lamp for indicating trigger and low-battery alarm, (2) an On-Off power switch, (3) a snap connector for the tube from the oxygen source, and (4) an electric plug from the sensor.

The start of each inspiration is detected by a sensor (ie, a copper-constantan thermocouple fixed to a nasal cannula near its prongs). In the production model, two thermocouples in series are used to obtain better sensitivity (double voltage) and double security (in case of unilateral obstruction of the nose). The respiratory sensors are exposed to airflow in front of the patient's nostrils to monitor gas temperature changes caused by respiration. (The reference point is located away from the nostrils in the atmospheric air.) The small thermocouples, which are sensitive and reliable enough to detect the initial point of inspiration without hysteresis and have been shown to be quick in response and suitable for mass production, are disposable parts.

The controller consists of a pair of input leads from the sensor, a differential amplifier, a low-pass filter, peak-hold circuits, comparators, a flip-flop, a pulse generator, and a solenoid valve to control the oxygen flow. In the production model, the electronic circuit in the controller has been simplified to be more compact and to save battery power (Fig. 2). The sensor generates output voltage proportional to the instantaneous value of the gas temperature, which increases during the expiratory

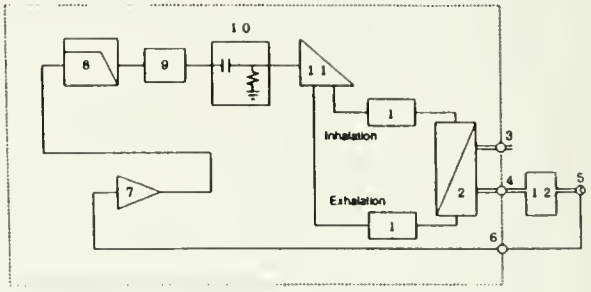

Fig. 2. Block diagram of a breath-synchronized device such as the Syncoxy—(1) pulse amplifier, (2) solenoid valve, (3) oxygen-flow inlet, (4) oxygen-flow outlet, (5) thermocouple sensors in nasal cannula, (6) leads input, (7) amplifiers, (8) low-pass filter, (9) automatic gain control, (10) differentiator circuit, (11) discriminator, and (12) optional humidifier.

phase and decreases during the inspiratory phase. An example of relationships among the inspiratory (I) and expiratory (E) phases of the respiratory cycle, electrical output from the sensor, the signal's amplification and modification, and the driving signals of the generator to open and close the solenoid valve are shown in Figure 3. Input voltage is amplified, filtered, and differentiated with respect to time, and thus initial points of the inspiratory and expiratory phases are detected. We decreased power consumption by using a trigger-type solenoid valve. An On-pulse of only 10-50 ms (DC 3 V) opens the valve and an Off-pulse of the same length closes the valve. Otherwise no electricity is used to operate the valve, thus extending the useful life of the batteries. An installed memory tip, when provided, allows prediction of the timing of the next phase for greater efficiency.[11] The controller converts respiration signals from analog to digital

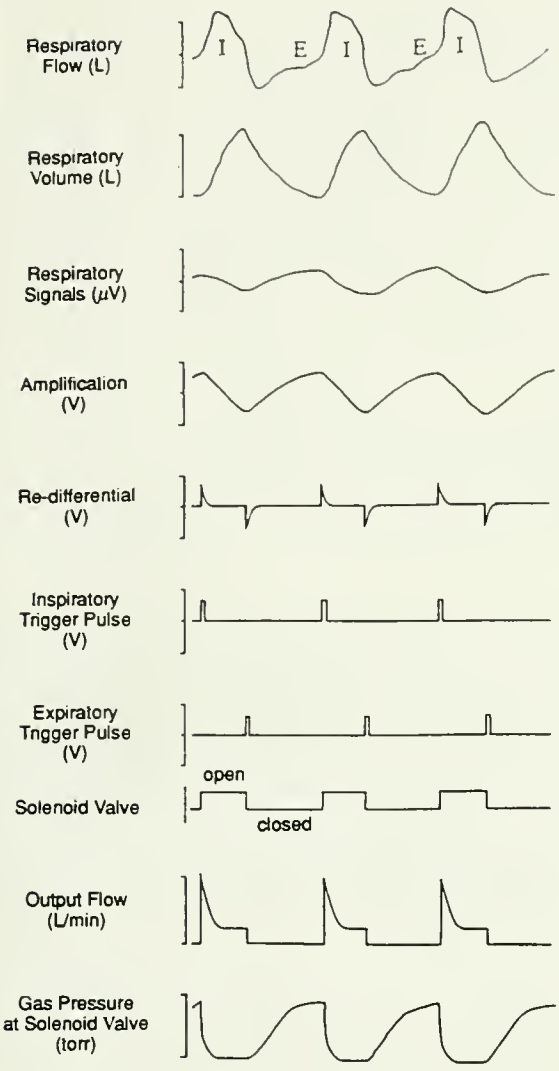

Fig. 3. Relationships among subject's inspiratory and expiratory flows, trigger pulses, and device output flows.

at a sampling rate of 100/s and differentiates them with respect to time for detecting gradients. Periods having negative gradients are recognized as inspiratory phases and periods with positive gradients as expiratory phases. These initial points are detected by the timing of polarity changes in the gradients or by the comparison of successive signal values by the two parallel comparators. Such timing pulses and reset pulses are repetitively generated at the maximum and minimum points of the detected signal to open and close the solenoid valve.

Gas is stored in a reservoir tank when the valve is closed and is then released with peak pressure to provide peak flow when the valve is opened. The pressure and flow patterns synchronized with each inspiration are shown in the two lower tracings of Figure 3.

Evaluation Method

Breath-Synchronized vs Continuous-Flow Administration in Normal Subjects

To compare the effects of breath-synchronized insufflation of oxygen to the effects of conventional continuous-flow insufflation at oxygen flows of 0.5, 1, 2, and 3 L/min and to compare the effects of the presence of reservoirs of 100, 200, and 400 mL in the breath-synchronized device, we monitored the transcutaneous partial pressure of oxygen (P_{tcO_2}) of 12 healthy adult volunteers by attaching a transcutaneous monitor to the forearm of each subject. We also analyzed samples of arterial blood drawn from the subjects during air breathing and during the breath-synchronized and continuous-flow insufflations of oxygen. Arterial samples obtained by conventional puncture and heparin technique were immediately analyzed in a calibrated automatic blood gas analyzer.

Breath-Synchronized vs Continuous-Flow Administration in COPD Patients

We measured arterial oxygen partial pressure (P_{aO_2}) in 24 and saturation by pulse oximeter (S_{pO_2}) in 21 COPD patients (convenience samples), respectively, while they were receiving oxygen at their prescribed flowrate by a continuous-flow device and by the SBSV.

Breath-Synchronized vs Conserver Administration

We determined the P_{tcO_2} resulting from the administration of oxygen at 0.5, 1, 2, and 3 L/min by the Pendant and Mustache Oxymizers, in 12 healthy adult male volunteers. The fractional concentration of inspired oxygen (F_{IO_2}) afforded by the continuous-flow device, the SBSV, and the two Oxymizers was also measured. We sampled by continuous suction (60 mL/min) through a fine PVC catheter (ID 1 mm) inserted into the hypo-

pharynx and a tiny polarographic electrode from a Hemox-analyzer into the reservoir of the Oxymizers to confirm a correlation between F_{IO_2} and F_{DO_2} (ie, the fractional concentration delivered by the device).

Administration by Two Electronic Breath-Synchronized Devices

We also observed the P_{tcO_2} resulting from the administration of oxygen at 1, 2, and 4 L/min via the Oxymatic on 8 healthy volunteers and compared it to P_{tcO_2} obtained with the SBSV at the same flows. In addition, we measured the delay time between the initial point of inspiration and the commencing of gas flow from the two devices on an oscilloscope in 10 healthy adult male volunteers. Gas consumption of the Oxymatic and of the SBSV required to produce equivalent P_{tcO_2} was also determined by electronic flowmetering devices.

Breath-Synchronized Valve with PSA Concentrator

We combined the SBSV with a 2-L/min pressure-swing-adsorption (PSA) oxygen concentrator and measured the oxygen concentration in the set output flowrates of 2-, 4-, and 7-L/min. The SBSV was connected to the outlet of the concentrator, and flow was adjusted by an installed flowmeter and measured by an anemometer.

Results

Breath-Synchronized vs Continuous-Flow Administration in Normal Subjects

The values for P_{aO_2} and P_{tcO_2} with the breath-synchronized and conventional continuous-flow devices at oxygen flows of 2 L/min are shown in Figure 4. The breath-synchronized devices with 100-mL and 200-mL reservoir tank capacities resulted in significantly higher P_{aO_2} and P_{tcO_2} than the continuous-flow devices ($p < 0.05$ by Student's *t* test).

The changes in P_{tcO_2} (mean, SD) at oxygen flows of 0.5, 1, 2, and 3 L/min obtained with the constant-flow device and with the breath-synchronized device with reservoirs of 100, 200, and 400 mL are shown in Figure 5. No significant differences were seen among different sizes of reservoir tank, while significant differences were seen between those and the constant flow, and also from air breathing as control ($p < 0.05$). Significant increases were also seen between 1 and 2 L/min flows in all groups ($p < 0.05$). In summary, synchronized insufflation supplied by central piped oxygen with or without external reservoirs of 100-400 mL almost doubles the increase in P_{tcO_2} seen with comparable flows during continuous-flow insufflation.

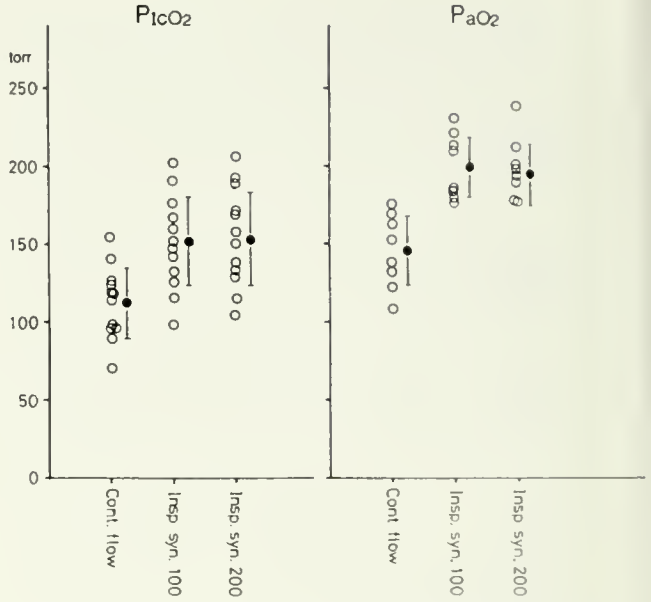

Fig. 4. Transcutaneous P_{O_2} (P_{tcO_2}) values in 12 normal volunteers with oxygen by continuous-flow device, breath-synchronized device with 100-mL reservoir, and breath-synchronized device with 200-mL reservoir (left) and arterial oxygen partial pressure (P_{aO_2}) values in 8 normal volunteers with oxygen by continuous-flow device, breath-synchronized device with 100-mL reservoir, and breath-synchronized device with 200-mL reservoir (right). Bars with closed circles indicate means with standard deviations.

Figure 6 shows one subject's P_{tcO_2} breathing air and breathing oxygen at three flowrates (0.5, 1.0, and 1.5 L/min). P_{tcO_2} was 79 torr by air, 87 torr with 0.5 L/min continuous-flow oxygen, and 99 torr with synchronization. With 1 L/min continuous flow P_{tcO_2} rose to 94, and with synchronization it rose to 123 torr. With 1.5 L/min, P_{tcO_2} rose to 109 torr with continuous flow and to 158 torr with synchronization.

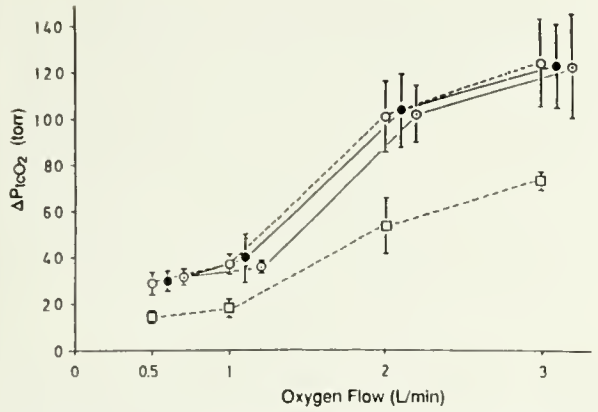

Fig. 5. Mean increases in P_{tcO_2} (over air-breathing level = 0 torr) in 12 normal volunteers, with oxygen flows of 0.5, 1, 2, and 3 L/min by continuous flow (□) and by synchronized breaths with reservoirs of 100 (○), 200 (●), and 400 mL (⊙).

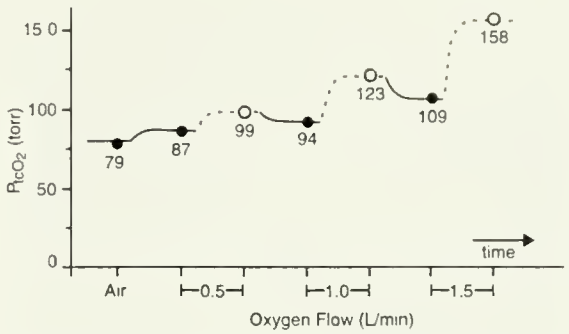

Fig. 6. Changes in P_{tcO_2} in a single healthy subject with changes in insufflation mode and oxygen flow (0, 0.5, 1.0, and 1.5 L/min). ● = continuous-flow oxygen; ○ = synchronized oxygen.

Breath-Synchronized vs Continuous-Flow Administration in COPD Patients

S_{pO_2} and P_{aO_2} (mean, SE) are shown in Figure 7. Both S_{pO_2} and P_{aO_2} were significantly increased with the SBSV (over the continuous-flow device) when flowrates were comparable ($p < 0.05$). A 30% reduction in flow with the SBSV turned on maintained oxygenation comparable to the continuous-flow device.

Breath-Synchronized vs Conserver Administration

The P_{tcO_2}s resulting from the administration of equivalent flows by the continuous-flow device, the SBSV, and the two conserver devices (Oxymizer Pendant and Mustache) and the F_{IO_2} provided by the four devices are shown in Figure 8. Inspiration-synchronized insufflation with the SBSV showed the highest P_{tcO_2} and F_{IO_2}, Oxymizer Pendant the next, Oxymizer Mustache the third, and continuous oxygen insufflation through a conventional nasal cannula the lowest.

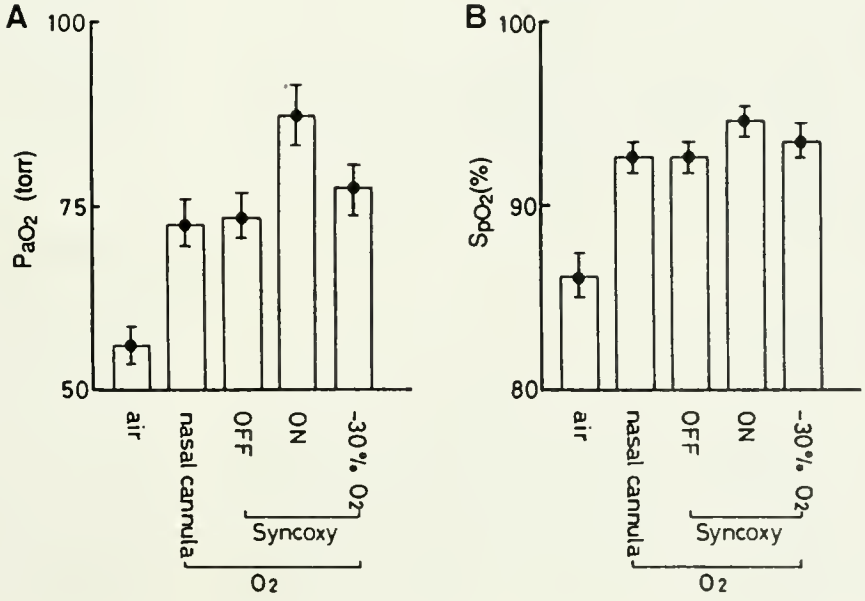

Fig. 7. A. P_{aO_2} values (mean, SE) in 24 COPD patients, breathing air and breathing oxygen by nasal cannula, by Syncoxy with solenoid valve off, with solenoid valve on, and with oxygen flow to Syncoxy reduced 30%. B. S_{pO_2} values (mean, SE) in 21 COPD patients, breathing air and breathing oxygen by nasal cannula, by Syncoxy with solenoid valve off, with solenoid valve on, and with oxygen flow to Syncoxy reduced 30%.

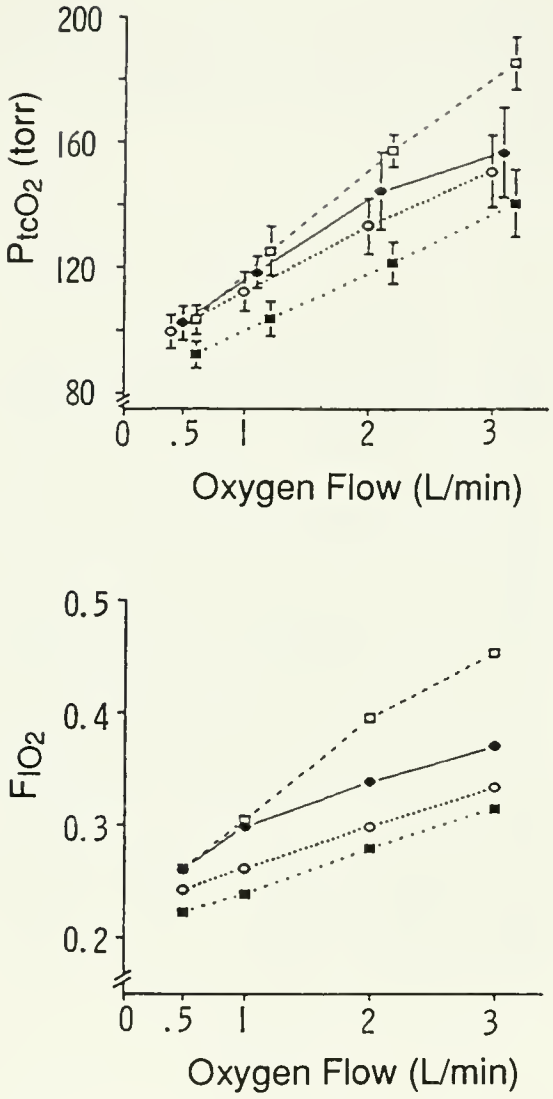

Fig. 8. Comparison of P_{tcO_2} (upper) attained in 12 healthy volunteers and F_{IO_2} values observed (lower), with continuous-flow (■), breath-synchronized insufflation by a Syncoxy device (□), breath-synchronized insufflation by Oxymizer Pendant (●), and breath-synchronized insufflation by Oxymizer Mustache (○).

Administration by Two Breath-Synchronized Devices

The P_{tcO_2}s measured in 8 healthy volunteers receiving oxygen at 3 flowrates via the SBSV and via the Oxymatic are shown in Figure 9. No significant differences were demonstrated including with continuous oxygen insufflation. However, the delay time of the SBSV was 0.2 s compared to a delay

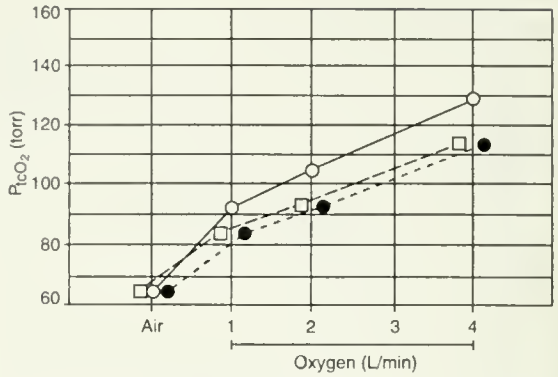

Fig. 9. Comparison of P_{tcO_2} attained in 8 healthy volunteers among 0, 1, 2, and 4 L/min oxygen continuous insufflation (□), Syncoxy (○), and Oxymatic (●) with same oxygen flowrates.

time of 0.9 for the Oxymatic. The oxygen consumption required to produce equal P_{tcO_2}s was 80% of the constant-flow device for the Oxymatic and 70% for the SBSV.

Breath-Synchronized Valve with PSA Concentrator

The oxygen concentrations provided by a 2-L/min concentrator gradually decreased with an increase in the output flowrate up to 7 L/min. However, with SBSV attached to the concentrator outlet, the oxygen concentration decreased less with the fewer number of openings associated with use of the synchronized valve. As shown in Figure 10, the concentration remained at almost 95% oxygen up to 7-L/min output flowrate when the valve opened and closed for 1 vs 4 intervals.

Discussion

Our results show that with the breath-synchronized device studied, the amount of oxygen provided through a nasal cannula can be reduced by 30% or more with the same oxygenation effects in healthy volunteers and in COPD patients. This is mainly due to elimination of the waste of oxygen associated with continued outflow during expiration. The peak outflow triggered by the very initial phase of inspiration and released as the synchronized valve opens appears to increase the initial F_{IO_2}. The storage of an adequate amount of oxy-

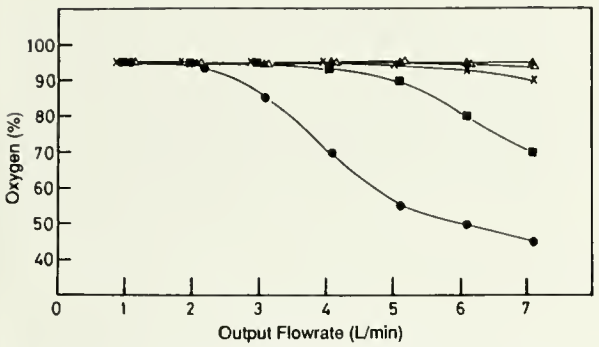

Fig. 10. Output flowrates and oxygen concentration characteristics of a PSA O_2 concentrator (100 V, 60 Hz, 22°C, relative humidity 30%) equipped with a breath-synchronized valve. Symbols represent ● = solenoid kept open, X = solenoid open 1 s, closed 2 s; Δ = 1 s open, 3 s closed; ■ = 2 s open, 2 s closed, ▲ = 1 s open, 4 s closed.

gen during expiration appears to be essential to increasing the oxygen utilization and oxygenation effects. However, when we used the breath-synchronized device with oxygen from the hospital's medical pipeline, no need for the extra reservoir upstream was seen—probably because the pipeline has the functional capacity to compensate for the reservoir. A PSA oxygen concentrator should have almost the same functional capacity as well, although we did not measure it exactly.

Reservoirs in Oxymizers are made of soft collapsible bag(s) in hard case(s) and are inserted in the nasal cannula near the patient end. During expiration such bags are automatically inflated by newly fed oxygen due to resistance against outflow. During the next inspiration, gas in the bag(s) is inhaled. Oxymizers are simple, convenient devices, but they provide less increase in oxygenation, especially at higher inspiratory flowrates—probably because their reservoirs (one 50-mL in the Pendant Oxymizer and two 10-mL in the Mustache) are not large enough.

A synchronized solenoid device triggered by pressure changes caused by respiration (Oxymatic) was also evaluated. The plastic case of this device (almost the same size as the Syncoxy) contains a pressure sensor, an electronic circuit, a 1.5-V dry battery, and a solenoid valve. The valve is kept open for 0.13 s during the initial inspiration and then is closed. Respiratory efforts of the patient are transmitted through a nasal cannula to the pressure sensor if they are intensive enough to trigger the valve, even though the system is open to the atmosphere. In our experience, pressure- and flow-generated signals are less easily transmitted in open systems than in closed systems like ventilator circuits. For this reason, we selected a thermocouple as the sensor.

Synchronized intermittent inhalation helps keep the nasal mucosa moist because the expired gas moistens the mucosa much as it does in physiologic respiration when flow is interrupted during expiration. In our experience, no patient complained of a dry nose despite the fact that we had eliminated the humidifiers during long applications of this device. However, the lack of complaints is not surprising because similar results have been reported with low-flow conventional cannulas,[12,13] and humidification may be no longer considered necessary with flows ≤ 4 L/ min.[14]

We believe that the combination of the SBSV with a PSA oxygen concentrator can improve concentrator performance and the oxygen concentration provided. In a PSA concentrator, a portion of the oxygen-enriched product gas must be used to purge nitrogen and water from the adsorption columns. With higher outflows, a less concentrated gas is produced because the volume of purging gas is relatively reduced and more nitrogen remains. During expiration with the SBSV in place, the outflow is suspended both in the adsorption columns and in the reservoir tank, and the stored gas is utilized for purging. This results in a lesser decrease in oxygen concentration at higher flowrates than when the SBSV is not used and should permit the use of a smaller, more portable concentrator.

Our study did not compare initial and replacement equipment and maintenance costs or costs of oxygen consumed.

Conclusions

We conclude from our results that use of the Syncoxy (1) can provide higher F_{IO_2}s and hence higher P_{aO_2}s than are possible at the same flow with continuous-flow devices, (2) can reduce the total amount of oxygen required to achieve a given effect, and (3) can enhance the performance of PSA-type oxygen concentrators, allowing higher oxygen concentrations to be delivered. In addition,

we believe it reasonable to state, although we have no objective data, that intermittent flow provided by the SBSV may be more comfortable than continuous flow for the patient.

PRODUCT SOURCES

Breath-Synchronized Oxygen-Delivery Devices:
Oxymatic, Chad Therapeutics Inc, Chatsworth CA
Syncoxy, Sanyo Denshi Co Ltd, Okayama, Japan

Reservoir-Type Oxygen-Conserving Devices:
Oxymizer Mustache, Chad Therapeutics Inc, Chatsworth CA
Oxymizer Pendant, Chad Therapeutics Inc, Chatsworth CA

Transcutaneous Monitor:
Hellige Oxymonitor SMS 361, Hellige GMBH, Freiberg, Breisgau, Germany

Oxygen Concentrator:
Sun-Fresh ME-951, Sumitomo Bakelite Co Ltd, Tokyo, Japan

Blood Gas Analyzer:
IL 1302, Instrumentation Laboratories Inc, Lexington MA

Oxygen Analyzer:
Model LC 700H, Toray Industries Inc, Tokyo, Japan

Pulse Oximeter:
Model N-100, Nellcor Inc, Hayward CA

Anemometer:
Anemomaster Model 6141, Nihon Kagaku Kogyo Co Ltd, Osaka, Japan

Flowmetering Devices:
Flowline SEF-51, STEC Inc, Kyoto, Japan
Model RF-2, Minato Medical Science Co Ltd, Osaka, Japan

Blood Gas Syringes:
P2-DO322, Terumo Corp, Tokyo, Japan

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An Alternative Method for Delivery of Ribavirin to Nonventilated Pediatric Patients

John L Cefaratt BS RRT and Evan A Steinberg MD

BACKGROUND: The need to administer ribavirin to pediatric patients with respiratory syncytial virus (RSV) pneumonia has generated major concerns related to the risk of drug exposure for caregivers and family members at the bedside. We sought to lower the possible exposure of those attending nonventilated patients to a time-weighted average of < 2.7 $\mu g/m^3$. METHOD & MATERIALS: In Phase 1, ribavirin was administered to a simulated patient within a hood placed in an oxygen tent; for Phase 2, a high-flow vacuum exhaust system was added; and for Phase 3, the mist generator of the oxygen tent was activated to saturate the environment of the tent external to the hood. Air samples were taken at three points: nearest, midpoint, and farthest from the source, for 5 to 6 hours each. Sample filters identified only by random numbers were sent for analysis by liquid chromatography. RESULTS: Time-weighted averages indicated a high exposure level for Phase 1 (123.0 $\mu g/m^3$), an 83.2% reduction for Phase 2 (20.7 $\mu g/m^3$), and a 98.3% reduction during Phase 3 (< 2.13 $\mu g/m^3$). CONCLUSION: We recommend that ribavirin administration to nonventilated patients incorporate a double-chambered hood and canopy, a high-flow vacuum exhaust pump, and a high-density mist generator. (Respir Care 1992;37:877-881.)

Introduction

Continuing controversy over possible risks to health care workers (HCW) from exposure to ribavirin during the treatment of young children with respiratory syncytial virus (RSV)[1-6] led us to investigate alternative means of drug delivery.

The age range of patients hospitalized for RSV infection (typically ≤ 3 years of age) limits delivery methods. In the past, attempts have been made to deliver ribavirin via aerosol face mask or simply by directed flow to the child's face. Because of the long administration period, neither of these methods has been found to be practical or tolerable for the patient or to allow predictable control over dissemination of medication to the hospital environment. Effective dual-canopy systems that offer less confinement to the patient yet limit the dissemination of aerosolized ribavirin have been described.[7-9]

Our study was designed to evaluate the effects of two modifications in delivery on ambient air levels of ribavirin. We began with an oxygen hood within an oxygen-tent canopy* to which first a high-flow vacuum exhaust system capable of evacuating up to 700 L/min (a prototype at the time of testing) and then a mist generator, or nebulizer (as

Mr Cefaratt is a Neo-Peds Respiratory Care Practitioner at Kaiser Permanente Los Angeles Medical Center, and holds a bachelor's degree in Environmental and Occupational Health. Dr Steinberg is Assistant Chief of Pediatrics at Kaiser Permanente Los Angeles Medical Center, and Associate Clinical Professor of Pediatrics at the University of Southern California School of Medicine—Los Angeles, California.

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The authors have no financial interest in the products described.

Reprints: Evan A Steinberg MD, 3rd Floor-Pediatrics, 4700 Sunset Blvd, Los Angeles CA 90027.

* Suppliers are identified in the Product Sources section at the end of the text.

found on many oxygen canopy tents), were added (Fig. 1). We hypothesized that because ribavirin is highly water soluble[10] suspended particles of the drug inside the tent would be captured by the high-density water aerosol particles and ultimately either rain out inside the tent or be exhausted through the vacuum system. We postulated that the combination of the high-flow vacuum exhaust system and the mist generator would provide maximum drug delivery to the patient and at the same time minimize exposure to all who entered the room. The end result should be an ambient level of ribavirin (ie, in the room) well below the safe level—a time-weighted average, or TWA, of < 2.7 $\mu g/m^3$—suggested by CalOSHA (the California Occupational Safety and Health Administration).[2]

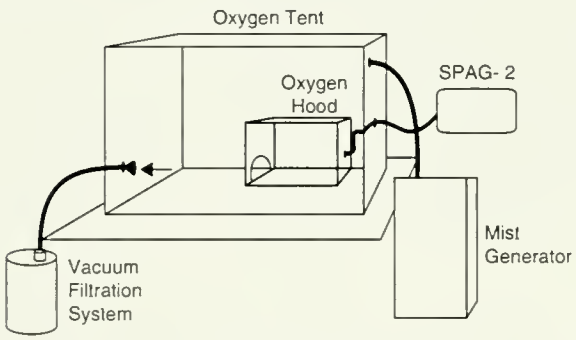

Fig. 1. Oxygen hood within an oxygen tent canopy for ribavirin administration.

Method and Materials

Equipment

The equipment utilized during the study included an oxygen tent with canopy, SPAG nebulizer (Series 6000) set according to hospital policy, compensated flowmeter set at 15 L/min, infant oxygen hood (without baffle sponge), rolled towel to simulate patient (for displacement at hood neck opening). samplers set at 2 L/min, digital calibrator, 12 special media filters (cellulose ester) pore size 0.45 μ, 1 liter of sterile water, and 3 vials of ribavirin mixed according to manufacturer's recommendations.

Calibration and Calculation

The SKC sample pumps were calibrated according to manufacturer's recommendations before and after each study test with an SKC Digital Calibrator. All pumps were set at sample rates of 2 L/min with the exact flows through each filter recorded both before and after each study test. A mean flowrate was then calculated. The mean flowrates were multiplied by the exact number of minutes for each test to provide the actual number of liters drawn for each sample. Liters were then converted to cubic meters (m^3) by the formula

$$\text{No. of liters}/1{,}000 = \text{No. of } m^3,$$

and then the result of the analysis reported in μg was divided by the sample volume in m^3 to yield a TWA in $\mu g/m^3$.

Methodology

Evaluations were conducted on two variations of a dual-canopy tent system. An oxygen hood inside of a CAM tent was used as the control. A 700 L/min vacuum exhaust system (supplier measured exhaust flow with standard intake anemometer) was first inserted at the foot of the tent and its effectiveness tested; subsequently, the mist generator of the CAM tent was activated in combination with the exhaust pump, and the effectiveness of the combination was tested. A patient model was placed in the hood to simulate an actual patient by displacing the hood opening. The SPAG-2 nebulizer was set up in the manner recommended by the manufacturer, with a total delivery flow of 15 L/min (7.5 L/min driving flow, 7.5 L/min to the drying chamber). The external flowmeter providing the source gas to the SPAG was attached to a blender to maintain consistency in the oxygen concentration delivered to the hood, and intermittent sampling of the concentration showed no change. All studies were conducted in an outvented patient room with the door remaining shut during the study as required by our institution's policy. The volume of the patient room was 1,581 ft^3, which with an outflow of 467.5 ft^3/min was provided with 17.7 air exchanges/hour (Fig. 2). Air samples were taken at three different locations during each phase of the evaluation. All samples were taken with SKC Samplers, with the cellulose ester filters in place. The pumps ran at 2 L/min and were placed in the same location for each phase of the evaluation—at distances of 2 feet, 6 feet, and 12 feet from the source

and at a height of 3-4 feet above the floor. Sampling ran for a minimum of 5 hours each. All filters were assigned a random 3-digit number, which was recorded by the investigator and used in analysis of results in an effort to limit possible bias.

Room size: 14' × 15 × 8.5' = 1,785 ft^3 – 204 ft^3 (bathroom) = 1,581 ft^3.

Air register: 9" × 17" = 153 in^2/144 in^2 = 1.06 ft^2, 80% open = (1.06 ft^2)(0.8) = 0.85 ft^2.

Average linear velocity = 550 ft/min.

Volumetric flow: 550 ft/min × 0.85 ft^2 = 467.5 ft^3/min.

Air turnover rate: (467.5 ft^3/min × 60 min/hr)/1,581 ft^3/change = 17.7 changes/hour.

Fig. 2. Calculations used in determining room air changes per hour.

In each phase of the study, the CAM tent was opened 3 times at spaced intervals for 15 minutes each to allow for normal patient care. In all cases, the standard practice was to shut off the SPAG nebulizer for a minimum of 5 minutes prior to the opening of the tent. During this time, supplemental oxygen and humidity were delivered to the patient hood tent via a one-way valve and T-adapter in-line to the SPAG delivery setup. The actual study design was set up to evaluate the dual-canopy tent system with two additional modifications: Phase 1—the dual-canopy oxygen tent system previously described; Phase 2—the addition of a high-flow vacuum exhaust system extracting up to 700 L/min; and Phase 3—employment of the mist generator of the CAM tent and the high-flow vacuum system. (The first control study had to be repeated because one of the air sample pumps failed approximately 2.5 hours into the study.)

Results

Time-weighted averages indicated a high exposure level for Phase 1 (123.0 $\mu g/m^3$), an 83.2% reduction for Phase 2 (20.7 $\mu g/m^3$), and a 98.3% reduction (< 2.13 $\mu g/m^3$) during Phase 3. Data from the three phases of the study are shown in Table 1; sample calculations are provided in Figure 3.

Total air sampled = (flow in L/min)(total time) = total liters.

Conversion of liters to cubic meters: (total liters)/(1,000 L/m^3) = m^3.

Time-weighted average (TWA) = total mass collected in μg/volume sampled in m^3 = TWA in $\mu g/m^3$.

Substituting the values for Sample KLA 405:
(1.95)(387) = 754.65 L.
754.65/1,000 = 0.755.
TWA = 15.47/0.755 = 20.5 $\mu g/m^3$.

Fig. 3. Calculations used in determining time-weighted-average exposure.

Discussion

Although the exact value of the samples collected while the vacuum unit and the mist generator were operative were too low to be accurately measured, it is safe to assume that the highest possible level is well below the safe standard accepted by CalOSHA. However, we do not advocate elimination of masks, goggles, or other protective devices if the HCW desires to wear them while in contact with ribavirin. These data suggest that the combination of a single high-volume vacuum exhaust system and the mist generator can keep the environmental levels within a safe range.

Our study was a bench study. However, we believe the results to be a valid predictor of clinical exposure if the protocol is followed, equipment is maintained, and the SPAG nebulizer is turned off for 5 minutes before the tent is opened.

Our method differs from the method of Torres et al[7] who employed wall suction (central system) with filters to scavenge the oxygen tent canopy and reported mean personal breathing zone concentrations of 43.81 ± 33.40 $\mu g/m^3$ of ribavirin. They suggest that because of the low level of absorption of ribavirin the levels that they observed were acceptable. We chose to use a rated vacuum pump because of possible fluctuations in the level of vacuum provided by the central suction system, and our goal was reduction to a TWA of < 2.7 $\mu g/m^3$. Our results may differ from theirs because of the difference in the vacuum source and because of differences in the placement of exhaust intake hoses. Kacmarek and Kratohvil[9] have described the ef-

Table 1. Measured and Calculated Values* for the Three Phases of a Bench Study of Ribavirin Scavenging Systems

Filter No.†	Flow (L/min)	Total Time (min)	Total Volume (L)	Volume Sampled (m^3)	Mass Collected (μg)	TWA‡ ($\mu g/m^3$)
Control Study (aborted because of pump failure)						
KLA 514	no data	378	no data	no data	7.56	no data
KLA 487	1.795	378	679	0.679	136.33	200.8
KLA 631	2.07	378	782	0.782	147.93	189.2
Control Study (complete)						
KLA 574	1.915	305	584	0.584	76.34	130.7
KLA 842	1.955	305	596	0.596	67.69	113.6
KLA711	1.85	305	564	0.564	70.34	124.7
Vacuum Exhaust (700 L/min)						
KLA 303	1.82	388	706	0.706	17.50	24.8
KLA 405	1.95	387	755	0.755	15.47	20.5
KLA 118	1.995	387	772	0.772	12.99	16.8 ± 20%
Vacuum Exhaust (700 L/min) **Plus Water Aerosol**						
KLA 104	1.85	302	559	0.559	< 1.25	< 2.2
KLA 290	1.955	302	590	0.590	< 1.25	< 2.1
KLA 993	1.995	301	600	0.600	< 1.25	< 2.1

* Figure 3 illustrates the calculations used.

† Numbers were assigned from a random number table.

‡TWA = time-weighted average.

ficacy of 2 and 3 vacuum pumps used in conjunction with a double-enclosure system and have reported TWA of < 2.7 $\mu g/m^3$ with that system. We obtained similar results by combining the effects of nebulized water and a single vacuum pump.

Our study does nothing to resolve the controversy of what level of ribavirin can be considered safe for the caregiver who may be exposed for many hours over the RSV 'season.' However, it does provide an apparently effective alternative to the system described by Kaemarek and Kratohvil,[9] and has provided reassurance to bedside caregivers in our institution.

Conclusion

We are not advocating that precautions and protective devices be eliminated when a high-flow vacuum exhaust system is coupled with a mist generator, but we are saying that we believe that with such a system there is little basis for high levels of anxiety on the part of HCW who are providing care to nonventilated patients during ribavirin administration.

PRODUCT AND SERVICE SOURCES

Tents and Hoods:

Mist-O-Gen CAM tent with canopy and mist generator, CAM-3 Peds Mist Tent with H-12 Nebulizer, Mist-O-Gen Equipment Co, Lancaster PA

Oxygen hood, Model 10H, Shiley Inc, Irvine CA

Flowmeters and Blenders:

Classic flowmeter Model 0-16, Timeter Instrumentation Corp, Lancaster PA

Blender, Model 3500HL, Sechrist Industries, Anaheim CA

Air Sampler, Components, and Calibrator:

SKC Aircheck sampler, Model 224-43XR, SKC West, Fullerton CA

Accuflow digital calibrator, Model 712, SKC West, Fullerton CA

Cellulose ester special media filters, pore size 0.45 μ, Millipore Filter Cassettes, SKC West, Fullerton CA

Nebulizer:

SPAG-2, Series 6000, ICN Pharmaceuticals, Costa Mesa CA

Medications:

Ribavirin (Virazole), ICN Pharmaceuticals Inc, Costa Mesa CA

Liquid Chromatography:

Assayed HPLC, ICN Pharmaceuticals Inc, Costa Mesa CA

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AARC Clinical Practice Guideline

Patient-Ventilator System Checks

MV-SC 1.0 PROCEDURE:

Patient-Ventilator System Check

MV-SC 2.0 DESCRIPTION:

2.1 A patient-ventilator system check is a documented evaluation of a mechanical ventilator and of the patient's response to mechanical ventilatory support. This procedure is often referred to simply as a ventilator check.

2.2 Objectives:

2.2.1 To evaluate and document the patient's response to mechanical ventilation at the time that the check is performed

2.2.2 To assure and document the proper operation of the mechanical ventilator

2.2.3 To verify and document that the ventilator is functioning and is properly connected to the patient

2.2.4 To verify and document that appropriate alarms are activated[1]

2.2.5 To verify and document that inspired gas is properly heated and humidified[1]

2.2.6 To verify and document that inspired oxygen concentration is measured with every change in F_{IO_2} or, at least, every 24 hours.[1] (Although retinopathy of prematurity, or ROP, is thought to be of multifactorial etiology,[2-4] the association between ROP and duration of exposure to high arterial oxygen levels suggests that continuous measurement of F_{IO_2} for infants at risk is warranted.[5])

2.2.7 To verify and document that ventilator settings comply with physician orders[1,6,7]

2.3 All data relevant to the patient-ventilator system check must be recorded on the appropriate hospital form(s) at the time of performance, must be included as an official part of the patient's medical record,[1,6,7] and include observations indicative of the ventilator's operation at the time of the check *(except where "should" is used)*:. Observations should include but are not limited to

2.3.1 observation that the ventilator is turned on and that the patient circuit is securely attached;[8]

2.3.2 documentation that an operational verification procedure (OVP, as described in the appropriate department's policy and procedure manual) was performed prior to or at the time that the ventilator was first applied to the patient;[1]

2.3.2.1 An OVP may be accomplished manually by occluding the patient connection and observing airway pressure rise on a pressure monitor or may be a self-test performed by the ventilator to assure proper internal function.

2.3.2.2 OVP should be performed at the bedside just prior to connection to the patient after the patient circuit has been changed or disassembled for any reason.

2.3.3 documentation that an alarm for airway disconnection is functional and is properly set;

2.3.4 documentation of measured F_{DO_2} (fractional concentration of oxygen delivered) with an appropriately calibrated analyzer;

2.3.5 documentation of measured inspired gas temperature, if applicable;

2.4 Patient-ventilator system checks must include patient information and observations

indicative of the ventilator's settings at the time of the check. *Observations should include but are not limited to*

2.4.1 Patient name
2.4.2 Patient hospital number
2.4.3 Diagnosis
2.4.4 Endotracheal or tracheostomy tube size and position
2.4.5 Documentation of time of last patient circuit change
2.4.6 Date of patient-ventilator system check
2.4.7 Time of patient-ventilator system check
2.4.8 Current ventilator settings
2.4.8.1 F_{DO_2} set and humidifier temperature setting (when applicable)
2.4.8.2 Mode of ventilation
2.4.8.3 Set ventilator frequency
2.4.8.4 Peak, mean, and baseline airway pressures and presence of auto-PEEP (if applicable)
2.4.8.5 Set peak inspiratory pressure limit and pressure support level, if applicable
2.4.8.6 Set tidal volume (if applicable)
2.4.8.7 Delivered tidal volume (measured or calculated)
2.4.8.8 Set sigh variables (if applicable)
2.4.8.9 Set minute ventilation (if applicable)
2.4.8.10 Set minimum mandatory minute ventilation (if applicable)
2.4.8.11 Set inspiratory flowrate and waveform (if applicable)
2.4.8.12 Set continuous flowrate (for IMV mode, if applicable)
2.4.8.13 Set I-E ratio, percent inspiration, or inspiratory and expiratory times
2.4.8.14 Set sensitivity threshold (if applicable)
2.4.9 Documentation of alarm settings and activation of appropriate alarms
2.4.10 A description of any instance of equipment failure
2.4.11 Signature of person performing patient-ventilator system check (including credentials) or initials (depending on state law and/or hospital policy)

2.5 Documentation of order from physician (or other authorized person) for mechanical ventilator settings—orders *should include at least one and preferably both of the following*:
2.5.1 Desired range for P_{aCO_2}, P_{tcCO_2}, and/or desired range for P_{aO_2}, S_{pO_2}, P_{tcO_2}, or S_{aO_2};
2.5.2 Ventilator variables to initiate or manipulate in order to achieve desired blood gas results (eg, mode, tidal volume, airway pressures, ventilatory frequency, or F_{DO_2}).

2.6 Patient-ventilator system checks must include, in brief narrative form, clinical observations indicative of the patient's response to mechanical ventilation at the time of the check. *Clinical observations should include but are not limited to an evaluation of*
2.6.1 breath sounds;
2.6.2 spontaneous respiratory rate, volume, and pattern;
2.6.3 chest motion;
2.6.4 pallor, skin color;
2.6.5 patient's level of consciousness or remarks;
2.6.6 endotracheal-tube cuff pressure and apparent stability and position of the tube
2.6.7 secretions;
2.6.8 condition of ancillary equipment (eg, chest tube apparatus and manual resuscitator);
2.6.9 results of bedside pulmonary function evaluations;
2.6.10 untoward effects of disconnection from ventilator during bedside procedures;
2.6.11 documentation of oxygenation and ventilation status (eg, arterial blood gas results, exhaled P_{CO_2} measurements, and transcutaneous saturation or transcutaneous blood gas measurements).
2.6.12 documentation of patient-ventilator synchrony during assisted or supported breaths

MV-SC 3.0 SETTINGS:

This guideline pertains to the in-hospital critical care setting.

MV-SC 4.0 INDICATIONS:

A patient-ventilator system check must be performed on a scheduled basis (which is institution-specific) for any patient requiring mechanical ventilation for life support. In addition, a check should be performed

4.1 prior to obtaining blood samples for analysis of blood gases and pH;
4.2 prior to obtaining hemodynamic or bedside pulmonary function data;
4.3 following any change in ventilator settings;
4.4 as soon as possible following an acute deterioration of the patient's condition (this may or may not be heralded by a violation of ventilator-alarm thresholds);
4.5 any time that ventilator performance is questionable.[9]

MV-SC 5.0 CONTRAINDICATIONS:

There are no absolute contraindications to performance of a patient-ventilator system check. If disruption of PEEP or F_{DO_2} results in hypoxemia, bradycardia, or hypotension, portions of the check requiring disconnection of the patient from the ventilator may be contraindicated.[10,11]

MV-SC 6.0 HAZARDS/COMPLICATIONS:

6.1 Disconnecting the patient from the ventilator during a patient-ventilator system check may result in hypoventilation, hypoxemia, bradycardia, and/or hypotension.[10,11]
6.2 Prior to disconnection, preoxygenation and hyperventilation may minimize these complications.[12-19]
6.3 When disconnected from the patient, some ventilators generate a high flow through the patient circuit that may aerosolize contaminated condensate, putting both the patient and clinician at risk for nosocomial infection.[20]

MV-SC 7.0 LIMITATIONS OF PROCEDURE/ VALIDATION OF RESULTS:

Measurements of volumes and inspired oxygen concentration are affected by the accuracy and reproducibility of the monitoring instruments.

7.1 Volume monitoring devices should be calibrated at regular intervals. Volume monitoring accuracy should be ±10% of the measured volume.[21]
7.2 Oxygen analyzers should be calibrated at regular intervals. Oxygen analyzer accuracy should be ±3% of actual concentration.

MV-SC 8.0 ASSESSMENT OF NEED:

Because of the complexity of mechanical ventilators and the large number of factors that can adversely affect patient-ventilator interaction, routine checks of patient-ventilator system performance are mandatory.

MV-SC 9.0 ASSESSMENT OF OUTCOME:

Routine patient-ventilator system checks should prevent untoward incidents, warn of impending events, and assure that proper ventilator settings, according to physician's order, are maintained.

MV-SC 10.0 RESOURCES:

10.1 Equipment: Appropriate equipment should be available to perform the patient-ventilator system check. *Such equipment should include but is not limited to*

10.1.1 stethoscope;
10.1.2 oxygen analyzer;
10.1.3 volume monitor (if applicable);
10.1.4 pressure monitor (if applicable);
10.1.5 supplies necessary for observing Universal Precautions.[21]

10.2 Personnel: Mechanical ventilation is a complex task that requires the caregiver to understand the technical components of the ventilator, the pathophysiology of the respiratory system, and patient-ventilator interaction. Persons performing checks should hold a recognized and relevant credential (eg, CRTT, RRT, RN), should be trained in and have demonstrated ability in

10.2.1 the technical setup and operation of the mechanical ventilator;
10.2.2 cardiopulmonary physiology and pathophysiology;

10.2.3 interpretation of the results of arterial blood gas analysis;
10.2.4 assessment of patient need for and adverse reaction to the procedure;
10.2.5 appropriate response to adverse reactions;
10.2.6 application of Universal Precautions.

MV-SC 11.0 MONITORING:

In order to assure that patient-ventilator system checks are being performed according to these guidelines, an indicator should be created to monitor this activity as part of the appropriate department's quality improvement program. *Specific criteria for the indicator should include at least items 2.4 and 12.0 of this guideline.*

MV-SC 12.0 FREQUENCY:

A patient-ventilator system check should be performed at regularly scheduled intervals and

12.1 following any change in ventilator settings;
12.2 prior to obtaining blood gas samples;
12.3 prior to obtaining hemodynamic or pulmonary function data;
12.4 as soon as possible following an acute deterioration of the patient's condition, particularly when this occurs after violation of a ventilator alarm threshold.

MV-SC 13.0 INFECTION CONTROL ISSUES:

13.1 Condensation from the patient circuit should be considered infectious waste and disposed of according to hospital policy.
13.2 The patient circuit should be changed at regular scheduled intervals according to hospital policy.
13.3 Universal Precautions should be observed during the patient-ventilator system check.[22]

Mechanical Ventilation Guidelines Committee:

Richard D Branson RRT, Chairman, Cincinnati OH
Robert S Campbell RRT, Cincinnati OH
Robert L Chatburn RRT, Cleveland OH
Jack Covington RRT, San Francisco CA

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AARC Clinical Practice Guideline

Humidification during Mechanical Ventilation

HMV 1.0 PROCEDURE:

The addition of heat and moisture to inspired gases delivered to the patient during mechanical ventilatory support via an artificial airway

HMV 2.0 DESCRIPTION/DEFINITION:

When the upper airway is bypassed, humidification during mechanical ventilation is necessary to prevent hypothermia, inspissation of airway secretions, destruction of airway epithelium, and atelectasis.[1-7] This may be accomplished using either a heated humidifier or a heat and moisture exchanger (HME). (HMEs are also known as hygroscopic condenser humidifiers, or artificial noses). The chosen device should provide a minimum of 30 mg H_2O/L of delivered gas at 30°C.[8-10] Heated humidifiers operate actively to increase the heat and water vapor content of inspired gas.[11-14] HMEs operate passively by storing heat and moisture from the patient's exhaled gas and releasing it to the inhaled gas.[15-25]

HMV 3.0 SETTINGS:

3.1 Critical care
3.2 Acute care inpatient
3.3 Extended care and skilled nursing facility
3.4 Home care
3.5 Prolonged transport

HMV 4.0 INDICATIONS:

Humidification of inspired gas during mechanical ventilation is mandatory when an endotracheal or tracheostomy tube is present.[1-7]

HMV 5.0 CONTRAINDICATIONS:

There are no contraindications to providing physiologic conditioning of inspired gas during mechanical ventilation. An HME is contraindicated under some circumstances.

5.1 Use of an HME is contraindicated for patients with thick, copious, or bloody secretions.[8,26-28]
5.2 Use of an HME is contraindicated for patients with an expired tidal volume less than 70% of the delivered tidal volume (eg, those with large bronchopleurocutaneous fistulas or incompetent or absent endotracheal tube cuffs).[15-25]
5.3 Use of an HME is contraindicated for patients with body temperatures less than 32°C.[8,29]
5.4 Use of an HME may be contraindicated for patients with high spontaneous minute volumes (> 10 L/min).[8,26,29]
5.5 An HME must be removed from the patient circuit during aerosol treatments when the nebulizer is placed in the patient circuit.[8,29]

HMV 6.0 HAZARDS/COMPLICATIONS:

Hazards and complications associated with the use of humidification devices include

6.1 potential for electrical shock—heated humidifiers;[11-14]
6.2 hypothermia—HME or heated humidifiers; hyperthermia—heated humidifiers;[11-14]
6.3 thermal injury to the airway from heated humidifiers;[30] burns to the patient and tubing meltdown if heated-wire circuits are covered or circuits and humidifiers are incompatible;

6.4 underhydration and impaction of mucus secretions—HME or heated humidifiers;[1-7]

6.5 hypoventilation and/or alveolar gas trapping due to mucus plugging of airways—HME or heated humidifier;[1-7]

6.6 possible increased resistive work of breathing due to mucus plugging of airways—HME or heated humidifiers;[1-7]

6.7 possible increased resistive work of breathing through the humidifier—HME or heated humidifiers;[31-34]

6.8 possible hypoventilation due to increased dead space—HME;[8,15-25,26-30]

6.9 inadvertent overfilling resulting in unintentional tracheal lavage—heated reservoir humidifiers;[35]

6.10 the fact that when disconnected from the patient, some ventilators generate a high flow through the patient circuit that may aerosolize contaminated condensate, putting both the patient and clinician at risk for nosocomial infection—heated humidifiers;[35]

6.11 potential for burns to caregivers from hot metal—heated humidifiers;

6.12 inadvertent tracheal lavage from pooled condensate in patient circuit—heated humidifiers;[35]

6.13 elevated airway pressures due to pooled condensation—heated humidifiers;

6.14 patient-ventilator dysynchrony and improper ventilator performance due to pooled condensation in the circuit—heated humidifiers;

6.15 ineffective low-pressure alarm during disconnection due to resistance through HME.[36]

HMV 7.0 LIMITATIONS OF METHODS:

7.1 Insufficient heat and humidification can occur with some HME devices, resulting in complications noted in HMV 6.0.[8,15-28]

7.2 Insufficient heat and humidification can occur with heated humidifiers and result in complications noted in HMV 6.0 when

7.2.1 improper temperature settings are selected.

7.2.2 water level in the humidifier falls below manufacturer's suggested level.[37]

7.3 The HME selected should be appropriate to the patient's size and tidal volume.

HMV 8.0 ASSESSMENT OF NEED:

Humidification is needed by all patients requiring mechanical ventilation via an artificial airway. Conditioning of inspired gases should be instituted using either an HME or a heated humidifier.

8.1 HMEs are better suited for short-term use (≤ 96 hours) and during transport.[8,29]

8.2 Heated humidifiers should be used for patients requiring long-term mechanical ventilation (> 96 hours) or for patients who exhibit contraindications for HME use.[8,29]

HMV 9.0 ASSESSMENT OF OUTCOME:

Humidification is assumed to be appropriate if, on regular careful inspection, the patient exhibits none of the hazards or complications listed in HMV 6.0.

HMV 10.0 RESOURCES:

10.1 Equipment: Appropriate equipment should be available to provide for adequate humidification of the inspired gas. Such equipment may include but is not limited to

10.1.1 humidification device;

10.1.2 a system to monitor inspired gas temperature and to alarm when the temperature falls outside a preset range (for heated humidifier);

10.1.3 sterile water for heated humidifiers;

10.1.4 equipment necessary to comply with Universal Precautions.

10.2 Humidifier performance specifications should be checked to assure adequate heating and humidification during expected peak inspiratory flowrate and minute ventilation delivered by the mechanical ventilator. Heated humidifiers selected for use should meet specifications of the American National Standards Institute.[9]

10.3 Personnel:

10.3.1 Level-1 personnel should possess

10.3.1.1 a complete understanding of the operation, maintenance, and troubleshooting of the ventilator, circuit, and humidifying device;

10.3.1.2 knowledge of and ability to implement Universal Precautions.

10.3.2 Level-II personnel should possess the abilities described in 10.3.1.1 and 10.3.1.2 and should also have

10.3.2.1 the ability to assess patient response to humidification;

10.3.2.2 the ability to recognize an adverse response to humidification;

10.3.2.3 the ability to appropriately respond to adverse events;

10.3.2.4 the ability to recommend modifications in humidification techniques as appropriate.

HMV 11.0 MONITORING:

The humidification device should be inspected visually during the patient-ventilator system check and condensate should be removed from the patient circuit as necessary. HMEs should be inspected and replaced if secretions have contaminated the insert or filter. The following variables should be recorded during equipment inspection:

11.1 Humidifier setting (temperature setting or numeric dial setting or both). During routine use on an intubated patient, a heated humidifier should be set to deliver an inspired gas temperature of 33 ± 2°C and should provide a minimum of 30 mg/L of water vapor.[8-10]

11.2 Inspired gas temperature. Temperature should be monitored as near the patient's airway opening as possible, if a heated humidifier is used.

11.2.1 Specific temperatures may vary with patient condition, but the inspiratory gas should not exceed 37°C at the airway threshold.

11.2.2 When a heated-wire patient circuit is used (to prevent condensation) on an infant, the temperature probe should be located outside of the incubator or away from the direct heat of the radiant warmer.[12]

11.3 Alarm settings (if applicable). High temperature alarm should be set no higher than 37°C, and the low temperature alarm should be set no lower than 30°C.[8,10]

11.4 Water level and function of automatic feed system (if applicable).

11.5 Quantity and consistency of secretions. Characteristics should be noted and recorded. When using an HME, if secretions become copious or appear increasingly tenacious, a heated humidifier should replace the HME.

HMV 12.0 FREQUENCY:

All patients with an artificial airway requiring mechanical ventilation should receive continuous humidification of inspired gases.

HMV 13.0 INFECTION CONTROL:

13.1 Reusable heated humidifiers should be subjected to high-level disinfection between patients.[35] Clean technique should be observed when manually filling the water reservoir. Sterile water should be used.

13.2 When using a closed, automatic feed system, the unused portion of water in the water feed reservoir remains sterile and need not be discarded when the patient circuit is changed. However, the water feed system should be designated for single patient use only.

13.3 Condensation from the patient circuit should be considered infectious waste and disposed of according to hospital policy using strict Universal Precautions.[35,38,39]

13.4 Because condensate is infectious waste, it should never be drained back into the humidifier reservoir.[35,38,39]

Mechanical Ventilation Guidelines Committee:

Richard D Branson RRT, Chairman, Cincinnati OH
Robert S Campbell RRT, Cincinnati OH
Robert L Chatburn RRT, Cleveland OH
Jack Covington RRT, San Francisco CA

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AARC Clinical Practice Guideline

Selection of Aerosol Delivery Device

AD 1.0 PROCEDURE:

Selection of a device for delivery of aerosol to the lower airways

AD 2.0 DESCRIPTION:

The selection of a device for administration of pharmacologically active aerosol to the lower airway. The device selected should produce particles with a mass median aerodynamic diameter (MMAD) of 2-5 microns.[1,2]

These devices include

- Metered dose inhalers (MDI)
- MDIs with accessory device (eg, spacer)
- Dry powder inhalers (DPI)
- Small volume nebulizers (SVN)
- Large volume nebulizers (LVN)
- Ultrasonic nebulizers (USN)

This guideline does not address bland aerosol administration and sputum induction.

AD 3.0 SETTING:

Aerosol therapy can be administered in a number of settings including hospital, clinic, extended care facility, and home.

AD 4.0 INDICATIONS:

The need to deliver—as an aerosol to the lower airways—a medication from one of the following drug classifications:

- Beta adrenergic agents
- Anticholinergic agents (antimuscarinics)
- Anti-inflammatory agents (eg, corticosteroids)
- Mediator-modifying compounds (eg, cromolyn sodium)
- Mucokinetics

The selection of a device for delivery of aerosol for parenchymal deposition (eg, antibiotics) will be addressed in another Guideline.

AD 5.0 CONTRAINDICATIONS:

5.1 No contraindications exist to the administration of aerosols by inhalation.

5.2 Contraindications related to the substances being delivered may exist. Consult the package insert for product-specific contraindications.

AD 6.0 HAZARDS/COMPLICATIONS:

6.1 Malfunction of device[3-5] and/or improper technique[6-12] may result in underdosing.

6.2 The potential exists for malfunction of device and/or improper technique (inappropriate patient use) to result in overdosing.

6.3 Complications of specific pharmacologic agent may occur.

6.4 Cardiotoxic effects of Freon have been reported as an idiosyncratic response that may be a problem with excessive use of MDI.[13-18]

6.5 Freon may affect the environment by its effect on the ozone layer.[19-21]

6.6 Repeated exposure to aerosols has been reported to produce asthmatic symptoms in some caregivers.[22]

AD 7.0 LIMITATIONS OF PROCEDURE OR DEVICE:

7.1 Only a small percent of output deposits in the airway (≤ 10%).[23-27]

7.2 Efficacy of the device is technique dependent (eg, coordination, breathing pattern, inspiratory hold).[6-9,28-32] The reader is referred to *Kacmarek RM, Hess D. The interface between patient and aerosol generator. Respir Care 1991;36:952-976* for detailed descriptions of optimal technique.

7.3 Efficacy of the device is design dependent (ie, output and particle size).[4,5,33]

7.4 Reduced deposition of aerosol to the lower airways is associated with the following and may require consideration of increased dose:

- **7.4.1** mechanical ventilation;[26,27,34-36]
- **7.4.2** artificial airways;[34-39]
- **7.4.3** airway caliber (eg, infants and children);[22,37-39]
- **7.4.4** severity of obstruction.[40-42]

7.5 Patient compliance[12]

7.6 Limitations of specific devices

- **7.6.1 Metered Dose Inhaler**:
  - **7.6.1.1** Environmental concerns(CFC)[20,21]
  - **7.6.1.2** Inadequate technique[6-8,28-32,43]
  - **7.6.1.3** Inadequate instruction[9-12]
- **7.6.2 MDI accessory device** (spacer or holding chamber):
  - **7.6.2.1** Adds to cost over MDI alone
  - **7.6.2.2** More bulky than MDI alone
- **7.6.3 Dry powder inhaler**:
  - **7.6.3.1** At the present time, patients must load each dose for most medications.
  - **7.6.3.2** Reduced inspiratory flow (< 60 L/min) can lead to reduced deposition.[44-46]
  - **7.6.3.3** Irritation to airway[33]
  - **7.6.3.4** Humidity may cause clumping of particles.
- **7.6.4 Small Volume Nebulizer**:
  - **7.6.4.1** Time- and labor-intensive
  - **7.6.4.2** Less portable
  - **7.6.4.3** Requires compressed-gas source or electricity
  - **7.6.4.4** Vulnerable to contamination[47-50]
  - **7.6.4.5** Lack of convenience may affect patient compliance
- **7.6.5 Large Volume Nebulizer:**
  - **7.6.5.1** Limited to acute and critical care setting
  - **7.6.5.2** Requires close monitoring
  - **7.6.5.3** Time- and cost-intensive
  - **7.6.5.4** Vulnerable to contamination[46-49]
  - **7.6.5.5** Reconcentration of solution may occur over long period of time due to evaporation by dry gas.
- **7.6.6 Ultrasonic Nebulizer**:
  - **7.6.6.1** Cost of device
  - **7.6.6.2** Mechanical reliability
  - **7.6.6.3** Requires electrical power source
  - **7.6.6.4** Vulnerable to contamination

AD 8.0 ASSESSMENT OF NEED:

8.1 Based on proven therapeutic efficacy,[25,51-60] variety of available medications, and cost-effectiveness[51,61-64] the MDI with accessory device should be the first method to consider for administration of aerosol to the airway.

8.2 Lack of availability of prescribed drug in MDI, dry powder, or solution form.

8.3 Inability of the patient to use device properly with coaching and instruction should lead to consideration of other devices.

8.4 Patient preference for a given device that meets therapeutic objectives should be honored.

8.5 When there is need for large doses, MDI, SVN, or LVN may be used. Clear superiority of any one method has not been established. Convenience and patient tolerance of procedure should be considered.

8.6 When spontaneous ventilation is inadequate (eg, as in kyphoscoliosis or neuromuscular disorders, exacerbation of severe bronchospasm with impending respiratory failure that does not respond to other forms of therapy), delivery by a positive pressure breathing device (IPPB) should be considered.[65-67]

AD 9.0 ASSESSMENT OF OUTCOME:

9.1 Proper technique applying device

9.2 Patient response to or compliance with procedure

9.3 Objectively measured improvement (eg, increased FEV_1 or peak flow)

AD 10.0 RESOURCES:

10.1 Equipment:

10.1.1 MDI—canister with actuator supplied by manufacturer; MDI accessory device that properly fits MDI mouthpiece or mask; adapters for specific circumstances (eg, tracheostomy)

10.1.2 Small volume nebulizer—gas source, tubing, flowmeter, and mouthpiece or mask

10.1.3 Large volume nebulizer—gas source, flowmeter, connecting tubing, and mouthpiece or mask

10.1.4 Mechanical ventilator—SVN or MDI, adapter in inspiratory line of circuit

10.1.5 IPPB machine (ie, pressure-limited ventilator)—nebulizer, gas source, connecting tubing, and mouthpiece or mask

10.1.6 Manual resuscitator—for 'bagging in' aerosol from MDI or SVN

10.2 Personnel:

10.2.1 Knowledge and skills at several levels are required to fully utilize and apply these devices.

10.2.1.1 Level II personnel provide initial assessments and care of the unstable patient:

10.2.1.1.1 utilizing proper technique for administration of MDI, accessory device, dry powder inhaler, SVN, LVN, USN; and SVN via IPPB;

10.2.1.1.2 practicing proper use, maintenance, and cleaning of equipment;

10.2.1.1.3 encouraging effective breathing patterns and coughing techniques;

10.2.1.1.4 modifying technique in response to adverse reactions;

10.2.1.1.5 modifying dosages and/or frequency as prescribed in response to severity of symptoms;

10.2.1.1.6 assessing patient condition and response to therapy;

10.2.1.1.7 performing auscultation and inspection and taking vital signs;

10.2.1.1.8 performing peak expiratory flowrate, spirometry, or ventilatory mechanics;

10.2.1.1.9 recognizing and responding to therapeutic and adverse responses and complications of medication and/or procedure;

10.2.1.1.10 understanding and complying with Universal Precautions.

10.2.1.2 The patient, family, or home caregiver; Level-I hospital personnel provide routine care of the patient:

10.2.1.2.1 preparing, measuring, and mixing medication;

10.2.1.2.2 demonstrating proper technique for administration of medication;

10.2.1.2.3 using equipment properly;

10.2.1.2.4 cleaning equipment;

10.2.1.2.5 encouraging effective breathing patterns and coughing techniques;

10.2.1.2.6 modifying technique in response to adverse reactions as instructed, with appropriate communication with physician or Level-II care provider;

10.2.1.2.7 modifying dosages and/or frequency as prescribed, with appropriate communication with physician, in response to severity of symptoms;

10.2.1.2.8 using the peak flowmeter properly and documenting results.

AD 11.0 MONITORING:

11.1 Performance of the device

11.2 Technique of device application

11.3 Assessment of patient response including changes in vital signs

AD 12.0 FREQUENCY:

12.1 Initiation of therapy after careful assessment of need (as outlined above)

12.2 The change from one type of device to another is based on a change in patient's condition or ability to use the specific device.

AD 13.0 INFECTION CONTROL:

13.1 Universal Precautions must be exercised for body substance isolation.[67]

13.2 SVN and LVN are for single patient use or should be subjected to high-level disinfection between patients.

13.3 Published data establishing a safe use-period for SVN and LVN are lacking; however they probably should be changed or subjected to high-level disinfection at approximately 24-hour intervals.

13.4 Medications:

13.4.1 Medications should be handled aseptically.

13.4.2 Tap water should not be used as the diluent.[69]

13.4.3 Medications from multidose sources in acute care facilities must be handled aseptically and discarded after 24 hours, unless manufacturer's recommendations specifically state that medications may be stored longer than 24 hours.

13.5 MDI accessory devices are for single patient use only. Cleaning of accessory devices is based on aesthetic criteria.

13.6 There are no documented concerns with contamination of medication in MDI canisters.

Aerosol Guidelines Committee:

Jon Nilsestuen PhD RRT, Chairman, Houston TX
Jim Fink MBA RRT, San Francisco CA
Theodore Witek Jr DrPH RPFT RRT, Ridgefield CT
James Volpe III MEd RRT, San Diego CA

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AARC Clinical Practice Guideline

Nasotracheal Suctioning

NTS 1.0 PROCEDURE:

Nasotracheal suctioning (NTS) is a component of bronchial hygiene therapy.

NTS 2.0 DESCRIPTION/DEFINITION:

NTS is intended to remove accumulated secretions, blood, vomitus, and other foreign material from the trachea that cannot be removed by the patient's spontaneous cough or other less invasive procedures. NTS has been used to avoid intubation that was solely intended for the removal of secretions.[1-4]

NTS refers to the insertion of a suction catheter through the nasal passage and pharynx into the trachea in order to aspirate accumulated secretions or foreign material.[3]

The clearance of secretions is accomplished by application of subatmospheric pressure.[5]

NTS 3.0 SETTINGS:

NTS is performed in a wide variety of settings, and this guideline applies to patients of all ages.

- **3.1** Critical care
- **3.2** Emergency room or department
- **3.3** Inpatient acute care
- **3.4** Extended care and skilled nursing facility care
- **3.5** Home care
- **3.6** Outpatient or ambulatory care

NTS 4.0 INDICATIONS:

The need to maintain a patent airway and remove secretions or foreign material from the trachea in the presence of

- **4.1** inability to clear secretions;[6]
- **4.2** audible evidence of secretions in the large/central airways that persist in spite of patient's best cough effort.[4,7,8-10]

NTS 5.0 CONTRAINDICATIONS:

Listed contraindications are relative unless marked as absolute.

- **5.1** Occluded nasal passages
- **5.2** Nasal bleeding
- **5.3** Epiglottitis or croup (absolute)
- **5.4** Acute head, facial, or neck injury
- **5.5** Coagulopathy or bleeding disorder[2]
- **5.6** Laryngospasm[2]
- **5.7** Irritable airway
- **5.8** Upper respiratory tract infection

NTS 6.0 HAZARDS/COMPLICATIONS:

- **6.1** Mechanical trauma[10-15]
  - **6.1.1** Laceration of nasal turbinates[5,7,16]
  - **6.1.2** Perforation of the pharynx[17]
  - **6.1.3** Nasal irritation/bleeding[16,18]
  - **6.1.4** Tracheitis
  - **6.1.5** Mucosal hemorrhage[13]
- **6.2** Hypoxia/hypoxemia[1,14,19-21]
- **6.3** Cardiac dysrhythmias/arrest[3,7,14,15]
- **6.4** Bradycardia[1,19,22-24]
- **6.5** Increase in blood pressure[1,19,21]
- **6.6** Hypotension[1,19]

6.7 Respiratory arrest[7]
6.8 Uncontrolled coughing[1,15,18]
6.9 Gagging/vomiting[18,25]
6.10 Laryngospasm[1,2,7]
6.11 Bronchoconstriction/bronchospasm[1,14,15]
6.12 Pain[18]
6.13 Nosocomial infection[15,16,23]
6.14 Atelectasis[5,14]
6.15 Misdirection of catheter[15,18]
6.16 Increased intracranial pressure (ICP)[21,26,27]
6.16.1 Intraventricular hemorrhage[21]
6.16.2 Exacerbation of cerebral edema

NTS 7.0 LIMITATIONS OF METHOD:

7.1 NTS is a blind, high-risk procedure with uncertain outcome.[18,24]
7.2 NTS should not be used to stimulate a cough.
7.3 Risks are increased in a combative or uncooperative patient.
7.4 Duration of application of subatmospheric pressure, or suction, should be limited to ≤ 15 seconds.[20,28]
7.5 Controversy exists concerning possible overuse of this procedure.[5,8,10]

NTS 8.0 ASSESSMENT OF NEED:

8.1 Personnel should auscultate chest for indications for NT suctioning.[1,29]
8.2 Personnel should assess effectiveness of cough.

NTS 9.0 ASSESSMENT OF OUTCOME:

9.1 Effectiveness of NTS should be reflected by improved breath sounds.
9.2 Effectiveness of NTS should be reflected by removal of secretions.

NTS 10.0 RESOURCES:

10.1 Equipment:
10.1.1 Vacuum source[1]
10.1.2 Calibrated, adjustable regulator[30]
10.1.3 Collection vessel and connecting tubing[1]
10.1.4 Sterile suction catheter of appropriate caliber[1,8,29]
10.1.5 Sterile disposable gloves[1,8]
10.1.6 Sterile water and cup[1,8]
10.1.7 Sterile normal saline—amount adequate for irrigation (5-10 mL for adults)[1,8,31]
10.1.8 Water-based lubricant[1,8]
10.1.9 Local anesthetic is sometimes used to reduce discomfort.[1]
10.1.10 Nasopharyngeal airway when frequent NTS is required[1,18,29]
10.1.11 Resuscitation bag with mask[1,23,29,31]

In the acute care setting, with initiation of NTS, or when working with the unstable patient, the following are recommended.

10.1.12 EKG monitor
10.1.13 Oxygen[1,20,28,32,33]
10.1.14 Personnel protective equipment for Universal Precautions[34,35]

10.2 Personnel:

10.2.1 Level I caregiver may be the provider of service *after* Level II personnel have established need by patient assessment and the first NTS episode has been completed. Level I personnel must demonstrate
10.2.1.1 knowledge of proper assembly and use of equipment;[7]
10.2.1.2 knowledge of upper airway anatomy and physiology;[7,23]
10.2.1.3 ability to recognize secretion retention on auscultation;[1]
10.2.1.4 ability to monitor vital signs and assess patient's condition and response to procedure;
10.2.1.5 ability to recognize and respond to adverse reactions and complications of procedures;
10.2.1.6 ability to employ technique of cardiopulmonary resuscitation when indicated;
10.2.1.7 ability to evaluate and document procedure effectiveness and patient response.

10.2.2 Level II provider initially assesses the patient, determines the need for NTS, and evaluates response to and effectiveness of first episode. **Level II** personnel *have all the skills of Level I providers plus*:

10.2.2.1 knowledge and understanding of patient's disease, goals, and limitation of NTS;[23]

10.2.2.2 recognition and understanding of basis of pathophysiology;

10.2.2.3 ability to perform initial treatment and be available to troubleshoot the procedure;

10.2.2.4 ability to modify techniques and equipment and take definitive action in response to adverse reaction;

10.2.2.5 ability to detect adverse reactions and avoid patient harm by employing techniques of cardiopulmonary resuscitation with mechanical airway adjuncts and bag-mask devices;

10.2.2.6 knowledge of basic EKG and dysrhythmia recognition;

10.2.2.7 knowledge of signs and symptoms of decreased cardiac output, oxygenation, and perfusion;

10.2.2.8 ability to teach Level I and lay personnel providing home care.

10.2.3 Home care should be provided by lay personnel trained and knowledgeable in

10.2.3.1 proper assembly and use of equipment;

10.2.3.2 correct positioning of patient;

10.2.3.3 proper suctioning technique;

10.2.3.4 assessment of patient response to procedure;

10.2.3.5 response to adverse reaction;

10.2.3.6 care and cleaning of equipment.

NTS 11.0 MONITORING:

The following should be monitored during and following the procedure.

11.1 Breath sounds
11.2 Skin color[36]
11.3 Breathing pattern and rate
11.4 Pulse rate, dysrhythmia, EKG if available
11.5 Color, consistency, and volume of secretions
11.6 Presence of bleeding or evidence of physical trauma
11.7 Subjective response including pain[25]
11.8 Cough
11.9 Oxygenation (pulse oximeter if available)
11.10 Intracranial pressure (ICP), if equipment is available

NTS 12.0 FREQUENCY:

Nasotracheal suctioning should be performed only when absolutely necessary and other methods to remove secretions from airway have failed.[4,5,8,10,29]

NTS 13.0 INFECTION CONTROL:

13.1 CDC Guidelines for Universal Precautions should be adhered to.[35]

13.2 All equipment and supplies should be appropriately disposed of or disinfected.

Bronchial Hygiene Guidelines Committee:

Lana Hilling RCP CRTT, Chairman, Concord CA
Eric Bakow MA RRT, Pittsburgh PA
James Fink MS RCP RRT, San Francisco CA
Chris Kelly BA RCP RRT, Oakland CA
Dennis Sobush MA PT, Milwaukee WI
Peter A Southorn MD, Rochester MN

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AARC Clinical Practice Guideline

Bronchial Provocation

BP 1.0 PROCEDURE:

Bronchial provocation test with methacholine and histamine

BP 2.0 DESCRIPTION/DEFINITION:

2.1 Bronchial provocation testing identifies and characterizes hyperresponsive airways by having the patient inhale an aerosolized bronchospastic agonist (such as methacholine or histamine for the purposes of this guideline). Results of pulmonary function tests (eg, spirometry, specific conductance) performed before and after the inhalation of increasing concentrations of the agonist are used to quantitate response. The procedure is also called bronchial challenge. This guideline applies to adults and children capable of adequately performing spirometry and cooperating during the course of the challenge.

2.2. A positive test is defined by one or more of the following:[1,2] a fall in $FEV_1 \geq 10\%$ in response to inhalation of the diluent; a decrease in FEV_1 from diluent FEV_1 value $\geq 20\%$ in response to inhalation of agonist; a fall in specific conductance of $\geq$ 35-40% from diluent value in response to inhalation of agonist. Changes in other spirometric values may also be evaluated.

BP 3.0 SETTING:

3.1 Hospital pulmonary function laboratory
3.2 Clinic or physician's office
3.3 Field site (eg, occupational setting or work place)

BP 4.0 INDICATIONS:

4.1 The need to diagnose or to confirm a diagnosis of airway hyperreactivity (asthma)[1,2]
4.2 The need to follow changes in hyperresponsiveness[1]
4.3 The need to document the severity of hyperresponsiveness[1,2]
4.4 The need to determine who is at risk in the military or workplace[1,2]
4.5 The need to establish a control or baseline prior to a series of environmental or occupational exposures[1]

BP 5.0 CONTRAINDICATIONS:

5.1 Relative contraindications are
- **5.1.1** existence of ventilatory impairment at the time of the proposed challenge.
  - **5.1.1.1** $FEV_1 < 80\%$ of previously recorded best value.[1]
  - **5.1.1.2** $FEV_1/FVC\% < 70\%$.[1]
  - **5.1.1.3** $sG_{aw} < 0.09\ s^{-1} \cdot cm\ H_2O^{-1}$.[1]
  - **5.1.1.4** $FEV_1 < 1.0$ L in adults.[2]
- **5.1.2** significant response to the diluent (> 10% fall in FEV_1 from baseline[1-3]).
- **5.1.3** upper- or lower-respiratory-tract infection within previous 6 weeks.[1]
- **5.1.4** specific antigen exposure within previous 1 week.[4]
- **5.1.5** exposure to high atmospheric pollution levels within previous 1 week.[1]
- **5.1.6** pregnancy (the effect on the fetus is unknown)[5]

5.2 subject's inability to perform acceptable spirometry (based on American Thoracic Society Guidelines[6]) at baseline and/or during post-diluent measurements (eg, variability in $FEV_1 > \pm 5\%$).

5.3 failure to withhold medications that may affect the bronchial reactivity test. Recommended periods for withholding medications are[1,2]

5.3.1 $Beta_2$-adrenergic aerosols—12 hours

5.3.2 Anticholinergic aerosols—12 hours

5.3.3 Disodium cromoglycate—8 hours

5.3.4 Oral $beta_2$-adrenergic agonists—12 hours

5.3.5 Theophyllines—48 hours

5.3.6 H_1-receptor antagonists—48 hours

5.3.7 Antihistamines—72-96 hours

5.3.8 Corticosteroids, inhaled or oral—have been shown to decrease hyperresponsiveness. Duration of effect is unknown but may be prolonged.

5.3.9 Beta blockers—may increase response, but duration of effect is unknown. Managing physician should be consulted before test is performed.

5.4 Other factors that may confound results include

5.4.1 Ingestion of cola drinks, chocolate, and other agents containing caffeine or theobromines;

5.4.2 Smoking;

5.4.3 Occupational exposure to antigens.

BP 6.0 HAZARDS/COMPLICATIONS:

The following untoward reactions may occur

6.1 Bronchoconstriction, hyperinflation, severe coughing;

6.2 Hazards associated with spirometry, such as dizziness, light-headedness, chest pain;[7]

6.3 Systemic hypotension and flushing from histamine;[8]

6.4 Possible exposure of technicians to provocative substances.

BP 7.0 LIMITATIONS OF METHOD VALIDATION OF RESULTS:

7.1 Limitations of pulmonary function testing used to quantitate response:

7.1.1 In some patients, spirometry may not be sensitive enough or specific enough to detect response, and other measurements such as airways resistance (R_{aw}) and/or specific conductance (sG_{aw}) may be indicated. Differences of opinion exist regarding which spirometric values best track response in particular airways.[1,2,4]

7.1.2 Deep inspiration taken while performing spirometry variably alters bronchial tone and may result in either bronchoconstriction or bronchodilatation.[9-11]

7.1.3 Poor patient effort during pulmonary function testing can produce false-positive results and make interpretation more difficult or impossible. Results from spirometry should be reproducible (ie, two FEV_1 measurements within 5% or two sG_{aw} measurements within 10%) after each dose.[6,7]

7.2 A limitation of the method is the variability due to the effects of various factors including medications, time of day, and differences in technique and equipment.[12]

7.3 Inconsistencies in technique and equipment can affect the amount of agonist reaching the airways and, thus, the subject's response—making meaningful interpretation difficult or impossible. Factors influencing response that must be controlled and held constant across testing include nebulizer output and particle size, volume inhaled, length of breath-hold, and inspiratory flowrate.[2,12]

7.4 If clinical suspicions are not confirmed by one test, additional tests (with the same or another agonist) may be indicated.

7.5 The final test report should document

7.5.1 adequacy of effort;

7.5.2 presence or absence of medications known to confound interpretation of results (Section 5.3);

7.5.3 presence or absence of other factors known to confound interpretation of results (Section 5.4);

7.5.4 clinical signs and symptoms and clinical appearance.

BP 8.0 ASSESSMENT OF NEED:

Need is established by documenting in a subject the presence of one or more of the listed indications.

BP 9.0 ASSESSMENT OF TEST QUALITY:

9.1 Factors that affect the size of the dose received and thus the response and its interpretation includs nebulizer output, particle size, inspiratory flowrate, lung volume at beginning of inspiration, and breath-hold time. These factors must be held constant across the testing procedure and from one test to another.

9.2 Excessive variability in measured values including a nonreproducible baseline (FEV_1 variation of more than 5% after repeated efforts) makes test results unacceptable.[6]

BP 10.0 RESOURCES:

10.1 Equipment:

10.1.1 Spirometers must meet or exceed ATS requirements[6] and be calibrated appropriately. All other equipment must be appropriately calibrated and maintained.

10.1.2 A high quality nebulizer with consistent output should be used to produce the aerosol. (Such a nebulizer may or may not be coupled to a dosimeter.) The particles produced by the nebulizer should have a mass median aerodynamic diameter (MMAD) of 2-5 microns.[1] If more than one nebulizer is used in the testing of a given subject, nebulizer output should be measured to assure a consistent dose. If output measurement is not possible, we recommend the use of the same nebulizer to deliver all concentrations to a given patient.

10.1.3 The gas powering the nebulizer and/or dosimeter should be at the correct driving pressure or flowrate (as specified by the manufacturer) and should be maintained at that pressure or flowrate consistently throughout the test.

10.1.4 Reagents:

10.1.4.1 The diluent in which the agonists are dissolved should be a 0.5% saline solution that also contains 0.275% $NaHCO_3$ and 0.4% phenol.[3] Other diluents may be recommended by the agonist manufacturer.

10.1.4.2 Various strategies have been described for challenging.[3,5,13-18] The range of doses is 0.02-25.0 mg/mL, generally given in doubling doses[1,3-5,12] (ie, 0.02 mg/mL, 0.04 mg/mL, 0.08 mL). The specific starting dose is chosen on the basis of the characteristics of the particular patient.

10.1.4.3 In general, methacholine solutions are stable for 4 months when stored at 4°C.[19,20] However, certain products may have their own recommendations that should be followed. The stability of histamine solutions is unknown but methacholine limits are probably appropriate.

10.1.4.4 Methacholine or histamine reagents should be prepared according to the manufacturer's recommendations by a pharmacist or other person trained in this procedure.

10.1.4.5 Reagents should be clearly labeled with dose and date prepared.

10.1.4.6 The test should be administered in a well-ventilated room. To our knowledge, no specifications for ventilation have been addressed in the literature. A filter to collect excess particles or an exhaust system to remove provocative material from the room may be desirable for the protection of other patients and the technician.

10.1.4.7 Oxygen, bronchodilators,[4] and resuscitation equipment should be readily available.[1,4]

10.1.4.8 The need for written consent should be determined within the specific institution.

10.2 Personnel:

10.2.1 Bronchial provocation should be performed under the direction of a physician trained in pulmonary function testing and experienced in bronchial provocation. Personnel performing the test should possess the qualifications of a Level-II person as described in the Spirometry Clinical Practice Guideline (10.2.1),[7] be experienced in patient assessment, knowledgeable about provocation testing and the

associated hazards, and be certified in CPR. One or more of the following credentials are recommended: CRTT, RRT, CPFT, RPFT.

10.2.2 During the testing procedure, a physician knowledgeable in provocation testing procedures and airway management should be readily available.[1]

BP 11.0 MONITORING:

11.1 The FEV_1 is the primary variable to be monitored, and the results of spirometry should meet all recommendations proposed by the ATS,[6] with reproducibility of FEV_1 and FVC within ±5% for each set of maneuvers associated with a given dose.

11.2 The test should be administered according to the specific protocol, with the number of breaths and the breathing pattern documented.

11.3 Periodic auscultation, pulse rate, and/or blood pressure may be monitored to assist in patient evaluation and test interpretation.[21,22]

11.4 In the case of a positive response to provocation (ie, > 20% fall in FEV_1), bronchodilator may be administered to speed recovery. Spirometry should be repeated after bronchodilator administration to ensure that ventilatory function has returned to near baseline.

BP 12.0 FREQUENCY:

12.1 The time that should be allowed to elapse to ensure that a previous challenge does not affect a later test is 90 minutes for histamine, 230 minutes for methacholine.[23]

12.2. When a test is to be repeated, medications, exposures, time of day, and nebulizer employed should be taken into consideration.

BP 13.0 INFECTION CONTROL:

13.1 Recommendations from the AARC Clinical Practice Guideline for Spirometry should be followed.[7]

13.2 Sterility of reagents should be maintained by proper storage and aseptic handling.

13.3 Nebulizer and mouthpiece should be cleaned and disinfected between patients.

Cardiopulmonary Diagnostics Guidelines Committee:

Kevin Shrake MA RRT, Chairman, Springfield IL
Susan Blonshine RPFT RRT, Lansing MI
Robert Brown BS RPFT RRT, Madison WI
Gregg Ruppel MA RPFT RRT, St Louis MO
Jack Wanger MBA RPFT RRT, Denver CO
Michael Kochansky CRTT RCPT RPFT, York PA

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AARC Clinical Practice Guideline

Exercise Testing for Evaluation of Hypoxemia and/or Desaturation

ET 1.0 PROCEDURE:

Exercise testing for evaluation of hypoxemia and/or desaturation

ET 2.0 DESCRIPTION/DEFINITION:

Exercise testing may be performed to determine the degree of oxygen desaturation and/or hypoxemia that occurs on exertion. Desaturation is defined as a valid decrease in arterial oxygen saturation as measured by CO-oximetry (S_{aO_2}) of 2% based on the reproducibility of HbO_2 measurement at ±1%[1] (although 4% is often used in the clinical literature[2,3]), an $S_{aO_2} < 88\%$,[4] and/or a $P_{aO_2} \leq 55$ torr.[4] Exercise testing may also be performed to optimize titration of supplemental oxygen for the correction of hypoxemia. This procedure should preferably be performed using a method that allows quantitation of work load and heart rate achieved (as % predicted). This evaluation can be incorporated into other more complex test protocols (eg, cardiac stress testing). Continuous noninvasive measurement of arterial oxyhemoglobin saturation by pulse oximetry can provide qualitative information and an approximation of oxyhemoglobin saturation, with a 4% decrease in S_{pO_2} considered significant,[5] but evaluation of desaturation on exertion requires analysis of arterial blood samples drawn with the subject at rest and at peak exercise.[6-12] Arterial blood specimens may be obtained by single puncture or by arterial cannulation.[13,14] Exercise testing performed with exhaled gas analysis is addressed in a separate guideline.

ET 3.0 SETTINGS:

Exercise testing may be performed by trained personnel in a variety of settings including hospital pulmonary function laboratories, cardiopulmonary exercise laboratories, clinics, and physicians' offices.

ET 4.0 INDICATIONS:

4.1 The need to assess and quantify the adequacy of arterial oxyhemoglobin saturation during exercise in patients who are clinically suspected of desaturation (eg, dyspnea on exertion, decreased D_{LCO}, decreased P_{aO_2} at rest)[2,4,7,15-17]

4.2 The need to quantitate the response to therapeutic intervention (eg, oxygen prescription, medications, smoking cessation)[2,4,7,18-20]

4.3 The need to titrate the optimal amount of supplemental oxygen to treat hypoxemia or desaturation during activity[4,7,20,21]

4.4 The need for preoperative assessment for lung resection or transplant[22]

4.5 The need to assess the degree of impairment for disability evaluation (eg, pneumoconiosis, asbestosis)[23]

ET 5.0 CONTRAINDICATIONS:

5.1 Absolute contraindications include

5.1.1 acute electrocardiographic changes of myocardial ischemia or serious cardiac dysrhythmias including bradydysrhythmias, tachydysrhythmias, sick sinus syndrome, and multifocal premature ventricular contractions (PVCs)—occasional PVCs are not a contraindication;[24-26]

5.1.2 unstable angina;[24,25]

5.1.3 recent myocardial infarction (within the previous 4 weeks) or myocarditis;[24,25]

5.1.4 aneurysm of the heart or aorta;[24,25]

5.1.5 uncontrolled systemic hypertension;[24,25]

5.1.6 acute thrombophlebitis or deep venous thrombosis;[24,25]

5.1.7 second- or third-degree heart block;[24,25]

5.1.7 second- or third-degree heart block;[24,25]

5.1.8 recent systemic or pulmonary embolus;[24,25]

5.1.9 acute pericarditis.[24,25]

5.2 Relative contraindications include

5.2.1 situations in which pulse oximetry may provide invalid data (eg, elevated HbCO, see Pulse Oximetry Guidelines[27]);

5.2.2 situations in which arterial puncture and/or arterial cannulation may be contraindicated;[28,29]

5.2.3 a non-compliant patient or one who is not capable of performing the test because of weakness, pain, fever, dyspnea, incoordination, or psychosis;[24,25]

5.2.4 severe pulmonary hypertension (cor pulmonale);[24,25]

5.2.5 known electrolyte disturbances (hypokalemia, hypomagnesemia);[24,25]

5.2.6 resting diastolic blood pressure > 110 torr or resting systolic blood pressure > 200 torr;[24,25]

5.2.7 neuromuscular, musculoskeletal, or rheumatoid disorders that are exacerbated by exercise;[24,25]

5.2.8 uncontrolled metabolic disease (eg, diabetes, thyrotoxicosis, or myxedema;[24,25]

5.2.9 S_{aO_2} or S_{pO_2} < 85% with the subject breathing room air;

5.2.10 untreated or unstable asthma.

ET 6.0 PRECAUTIONS AND/OR POSSIBLE COMPLICATIONS:

6.1 Indications for immediate termination of testing include

6.1.1 electrocardiographic abnormalities (eg, dangerous dysrhythmias, ventricular tachycardia, ST-T wave changes);[24,25]

6.1.2 a severe desaturation as indicated by an $S_{aO_2} \leq 80\%$ or $S_{pO_2} \leq 83\%$ (because a number of pulse oximeters have been found to overestimate S_{pO_2}[3,12,30,31] and/or a 10% fall from baseline values;

6.1.3 angina;[24,25]

6.1.4 hypotensive responses

6.1.4.1 a fall of > 20 torr in systolic pressure, occurring after the normal exercise rise;[32]

6.1.4.2 a fall in systolic blood pressure below the pre-exercise level.[32]

6.1.5 lightheadedness;[24,25]

6.1.6 request from patient to terminate test.

6.2 Abnormal responses that may require discontinuation of exercise include

6.2.1 a rise in systolic blood pressure to > 250 torr or of diastolic pressure to > 120 torr,[24,25] or a rise in systolic pressure of < 20 torr from resting level;

6.2.2 mental confusion or headache;[24,25]

6.2.3 cyanosis;[24,25]

6.2.4 nausea or vomiting;

6.2.5 muscle cramping.[24,25]

6.3 Hazards associated with arterial puncture, arterial cannulation, and pulse oximetry[27-29]

ET 7.0 LIMITATIONS OF PROCEDURE/ VALIDATION OF RESULTS:

7.1 Limitations

7.1.1 Equipment:

7.1.1.1 Due to possible limitations of pulse oximetry with exercise and at rest, measurements may read falsely low or falsely high and should be validated by comparison with baseline arterial samples analyzed by CO-oximetry.[27]

7.1.1.1.1 Only a limited number of pulse oximeters have been validated under exercise conditions with concurrent arterial blood gas analysis under exercise conditions in diseased subjects.[15]

7.1.1.1.2 Overestimation of oxygen saturation may occur with carboxyhemoglobin saturations greater than 4-4.9%.[2,33]

7.1.1.1.3 Decreasing accuracy in S_{pO_2} has been reported with desaturations to < 83%.[12]

7.1.1.1.4 Decreased perfusion with cardiovascular disease may result in false-positive results or no valid data.[10,34]

7.1.1.1.5 Reduced ear perfusion associated with heavy exercise has

7.1.1.1.6 Motion artifact may appear with exercise.[15,38]

7.1.1.1.7 Pulse oximeter response time may be inadequate to describe rapid changes in saturation.[7,15]

7.1.1.1.8 Skin pigmentation may affect pulse oximeter readings.[3]

7.1.1.1.9 Hemoglobin disorders may affect the accuracy of the pulse oximeter reading.[15] Important underestimation of arterial saturation may result with pulse oximetry in subjects with total hemoglobin levels of ≤ 8 g/dL.[39]

7.1.1.1.10 Pulse oximetry is less useful over the range in which large changes in P_{aO_2} are associated with small changes in S_{aO_2} (ie, $P_{aO_2} \geq 60$ torr).[15]

7.1.2 Additional limitations common to arterial sampling and analysis under resting conditions, should be considered.[28,29]

7.1.3 Patient cooperation level or physical condition may limit the subject's ability to exercise at a work load sufficient to evoke a response.[24,25]

7.1.4 Results of exercise testing in which work load is not quantified or in which cardiovascular variables are not adequately monitored (eg, free walking) have limited application.

7.2 Validation of results:

7.2.1 Arterial blood gas samples should be obtained at rest and at peak exercise.[9] Samples from single arterial punctures have been shown to be equivalent to samples drawn from indwelling cannulas.[14]

7.2.2 In the unlikely event that single puncture at peak exercise is unsuccessful in an uncannulated patient, a sample drawn within 10-15 seconds of the termination of exercise will suffice unless analysis shows a decrease from the resting values, in which case quantitation of desaturation requires a peak exercise sample obtained by cannula.[14]

7.2.3 Arterial blood gas results should be obtained according to the Guidelines for arterial blood gas sampling and for arterial blood gas analysis.[28,29]

7.2.4 Validity of pulse oximetry results is verified by comparison with the results of analysis by CO-oximetry,[27] preferably at rest and at end of exercise.

7.2.4.1 S_{pO_2} may be used to assess response to supplemental oxygen. If administration of supplemental oxygen does not improve a low S_{pO_2}, arterial blood analysis may be warranted.

7.2.4.2 Testing should be performed in compliance with the Pulse Oximetry Clinical Practice Guideline.[27]

ET 8.0 ASSESSMENT OF NEED:

Exercise testing for evaluation of hypoxemia and/or desaturation may be indicated for the following reasons:

8.1 the presence of a history and physical indicators suggesting hypoxemia and/or desaturation (eg, dyspnea, pulmonary disease);

8.2 the presence of abnormal diagnostic test results (eg, D_{LCO}, FEV_1, resting arterial blood gases including directly measured HbO_2, HbCO, and HbMet);

8.3 the need to titrate or adjust a therapy (eg, supplemental oxygen).

ET 9.0 ASSESSMENT OF TEST QUALITY:

9.1 Arterial blood gases and/or S_{pO_2} should confirm or rule out oxygen desaturation during exercise to validate the patient's clinical condition.

9.2 Documentation of results, therapeutic intervention (or lack of) and/or clinical decisions based on the exercise testing should be placed in the patient's medical record.

9.3 The exercise should have a symptom-limited or physiologic end point documented (eg, heart rate or onset of dyspnea).

ET 10.0 RESOURCES:

10.1 Equipment:

10.1.1 Treadmill, cycle ergometer, or equivalent equipment adaptable to patients

who may be severely limited (eg, low-speed treadmill, low-watt ergometer, arm crank).[24,25,40,41] Other forms of exercise may be utilized (stair climbing, step test, timed walking); however, such modes do not eliminate the necessity for adequate monitoring as described in Sections 7 and 9 and the necessity for adequate documentation of procedure and patient response

10.1.2 Arterial blood sampling equipment for single puncture or arterial cannulation and analyzers that have been properly calibrated and for which multilevel controls indicate proper function[28,29]

10.1.3 Pulse oximeter and related accessories (optional)[27]

10.1.4 Electrocardiographic monitor with the capacity to monitor heart rate to a predicted maximum and accurately display cardiac rhythm during exercise (multiple leads are preferred[24,25])

10.1.5 Resuscitation equipment including oxygen[24,25]

10.1.6 Access to cardiac arrest cart[24,25]

10.1.7 Blood pressure monitoring device, manual or automatic (if an automated system is used, a manual blood pressure cuff and stethoscope should be available as a backup)[24,25]

10.2 Background history and data:

10.2.1 Results of appropriate baseline diagnostic tests and patient history (eg, electrocardiogram, chest x-ray, and pulmonary function test results) should be available[24-26]

10.2.2 The need for written consent should be determined within the specific institution.[25,26]

10.3 Personnel:

10.3.1 The presence of a physician trained in exercise testing may be required depending on the patient condition and hospital policy.[24,25]

10.3.2 Personnel administering the test should possess experience and knowledge in exercise physiology and testing, including arterial blood gas sampling and analysis, cardiopulmonary resuscitation (BCLS certified), ECG abnormality recognition, oxygen therapy, blood pressure monitoring, and application and limitations of pulse oximeters.

ET 11.0 MONITORING:

11.1 Recommended monitoring equipment:

11.1.1 Electrocardiograph with strip recorder, preferably with multiple leads

11.1.2 Pulse oximeter (heart rate correlated with electrocardiograph if available)

11.1.3 Oxygen delivery devices with documented F_{DO_2}

11.1.4 Treadmill or cycle ergometer calibrated according to the manufacturer's recommendations, with periodic reverification

11.1.5 Blood gas sampling and analysis equipment[28,29]

11.2 Recommended patient monitors during testing:

11.2.1 Physical assessment (chest pain, leg cramps, color, perceived exertion, dyspnea)[24,25]

11.2.2 Respiratory rate[24,25]

11.2.3 Cooperation and effort level

11.2.4 Borg or modified Borg dyspnea scale

11.2.5 Blood gas sampling site and technique according to the Clinical Practice Guideline for blood gas sampling[28]

11.2.6 Heart rate, rhythm, and ST-T wave changes[24,25]

11.2.7 Blood pressure[24,25]

ET 12.0 FREQUENCY:

The frequency of testing depends on the patient's clinical condition and the need for changes in therapy. Exercise may be repeated for certification of supplemental oxygen needs.

ET 13.0 INFECTION CONTROL:

13.1 Recommendations from the Clinical Practice Guidelines for pulse oximetry, arterial blood sampling, and blood gas analysis should be followed.[27-29]

13.2 Disposable items are for single patient use.

13.3 Disposable electrodes should be used for electrocardiographic monitoring with Universal Precautions observed during patient skin preparation.

Cardiopulmonary Diagnostics Guidelines Committee:

Kevin Shrake MA RRT, Chairman, Springfield IL
Susan Blonshine RPFT RRT, Lansing MI
Robert Brown BS RPFT RRT, Madison WI
Gregg Ruppel MA RPFT RRT, St Louis MO
Jack Wanger MBA RPFT RRT, Denver CO
Michael Kochansky CRTT RCPT RPFT, York PA

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AARC Clinical Practice Guideline

Sampling for Arterial Blood Gas Analysis

ABS 1.0 PROCEDURE:

Sampling for arterial blood gas analysis

ABS 2.0 DESCRIPTION:

Blood is drawn anaerobically from a peripheral artery (radial, brachial, femoral, or dorsalis pedis) via a single percutaneous needle puncture, or from an indwelling arterial cannula or catheter for multiple samples.[1-8]

Either method provides a blood specimen for direct measurement of partial pressures of carbon dioxide (P_{aCO_2}) and oxygen (P_{aO_2}), hydrogen ion activity (pH), total hemoglobin (Hbtotal), oxyhemoglobin saturation (HbO_2), and the dyshemoglobins carboxyhemoglobin (COHb) and methemoglobin (MetHb).

ABS 3.0 SETTING:

Sampling may be performed by trained health care personnel[6-8] in a variety of settings including (but not limited to) hospitals, clinics, physician offices, extended care facilities, and the home.

ABS 4.0 INDICATIONS:

4.1 The need to evaluate the adequacy of ventilatory (P_{aCO_2}), acid-base (pH and P_{aCO_2}), and oxygenation (P_{aO_2} and S_{aO_2}) status, and the oxygen-carrying capacity of blood (P_{aO_2}, HbO_2, Hbtotal, and dyshemoglobins)[1,2,7,8]

4.2 The need to quantitate the patient's response to therapeutic intervention[1,2,5] and/or diagnostic evaluation (eg, oxygen therapy, exercise testing)[2]

4.3 The need to monitor severity and progression of a documented disease process[1,2]

ABS 5.0 CONTRAINDICATIONS:

Contraindications are absolute unless specified otherwise.

5.1 Negative results of a modified Allen test (collateral circulation test) are indicative of inadequate blood supply to the hand[1] and suggest the need to select another extremity as the site for puncture.

5.2 Arterial puncture should not be performed through a lesion or through or distal to a surgical shunt (eg, as in a dialysis patient). If there is evidence of infection or peripheral vascular disease involving the selected limb, an alternate site should be selected.

5.3 Agreement is lacking regarding the puncture sites associated with a lesser likelihood of complications; however, because of the need for monitoring the femoral puncture site for an extended period, femoral punctures should not be performed outside the hospital.

5.4 A coagulopathy or medium-to-high-dose anticoagulation therapy (eg, heparin or coumadin, streptokinase, and tissue plasminogen activator but not necessarily aspirin) may be a relative contraindication for arterial puncture.

ABS 6.0 HAZARDS/COMPLICATIONS:

6.1 Hematoma[1,2,7,8]

6.2 Arteriospasm[1,7,8]

6.3 Air or clotted-blood emboli[1-3,7,8]

6.4 Anaphylaxis from local anesthestic[7,8]

6.5 Introduction of contagion at sampling site and consequent infection in patient; introduction of contagion to sampler by inadvertent needle 'stick.'[1-3,7,8]

6.6 Hemorrhage[2,3]

6.7 Trauma to the vessel[1,3]
6.8 Arterial occlusion[2]
6.9 Vasovagal response
6.10 Pain

ABS 7.0 LIMITATIONS OF METHOD/VALIDATION OF RESULTS:

7.1 Limitations:

7.1.1 Artery may be inaccessible due to periarterial tissues (overlying muscle, connective tissue, or fat).[1]

7.1.2 Pulse may not be palpable.[7,8]

7.1.3 Arteriospasm may preclude collection despite successful introduction of needle into the artery.[7,8]

7.1.4 Arterial blood specimens withdrawn from the body only reflect the physiologic condition at the moment of sampling (eg, pain from the puncture itself may lead to hyperventilation with consequent changes in values).

7.1.5 Specimens drawn at peak exercise best reflect response to exercise; however, specimens drawn within 15 seconds or less of termination of exercise may be acceptable (otherwise results do not reflect ventilatory status during dynamic activities and may yield false-negatives for hypoxemic events).[9,10]

7.1.6 Specimens from mechanically ventilated patients with minimal pulmonary pathology adequately reflect the effects of oxygen concentration change 10 minutes after the change.[7,8,11] In spontaneously breathing patients, at least 20-30 minutes should elapse following oxygen concentration change (patients with obstructive defects and increased residual volumes may require the full 30 minutes or longer).

7.1.7 Specimens held at room temperature must be analyzed within 10-15 minutes of drawing; iced samples should be analyzed within 1 hour.[1] The P_{aO_2} of samples drawn from subjects with elevated white cell counts may decrease very rapidly. Immediate chilling is necessary.[12,13] Some dual-purpose electrolyte/blood gas analyzers stipulate immediate analysis without chilling because of possible elevations in potassium from chilling;[14] however, the accuracy of the blood gas results should not be affected by the chilling.

7.2 Validation of results

7.2.1 Sample must be obtained anaerobically and anticoagulated, with immediate expulsion of air bubbles. Sample should be immediately chilled or analyzed within 10-15 minutes if left at room temperature.[15]

7.2.2 When a sample is obtained, date, time, patient's body temperature, position, activity level, respiratory rate, sample site, results of Allen test, inspired oxygen concentration[4,6-8] or supplemental oxygen flow, and mode of supported ventilation[4] should be documented in the patient's medical record with the results of blood gas analysis.

7.2.3 Appropriate sample size depends on (1) the anticoagulant used, (2) the requirements of the specific analyzers to be used, and (3) the presence of a need for other assays.

7.2.3.1 If liquid heparin (sodium or lithium, 1,000 units/mL of blood) is used, excess heparin (all except that filling the dead space of the syringe and needle) should be expelled and a blood sample of 2-4 mL be drawn (liquid heparin dilutes the specimen and changes P_{CO_2} and P_{O_2} in direct relationship to the heparin volume).[16]

7.2.3.2 If lyophilized heparin is used, the minimum volume drawn depends on the design of the analyzers and the need for other assays.

7.2.3.3 If other assays are required (eg, electrolyte determination), the choice of anticoagulant and the volume of the blood sample should be guided by the analyzer manufacturer's recommendations.

ABS 8.0 ASSESSMENT OF NEED:

The following findings may assist the clinician in deciding whether arterial blood sampling is indicated:

8.1 History and physical indicators (eg, positive smoking history, recent onset of difficulty in breathing independent of activity level, trauma)[2]

8.2 Presence of other abnormal diagnostic tests or indices (ie, abnormal pulse oximetry reading, chest x-ray)[2]

8.3 Initiation of, administration of, or change in therapeutic modalities (eg, initiation, titration or discontinuance of supplemental oxygen or initiation of, changes in, or discontinuance of mechanical ventilation)[1,2,4,5]

8.4 Projected surgical interventions for patients at risk[5,17]

8.5 Projected enrollment in a pulmonary rehabilitation program

ABS 9.0 ASSESSMENT OF TEST QUALITY:

9.1 Sampling of arterial blood for any of the indications listed will be useful for patient management only if the sampling procedure is carried out according to an established, proven protocol. The validity of test results can be voided if any of the following occur:[1,6-8]

9.1.1 The sample is contaminated by air, improper anticoagulant or inappropriate anticoagulant concentration, flush solution (if sample is drawn from an indwelling catheter), or venous blood.[1,2,4,6-8]

9.1.2 The sample clots because of improper anticoagulation of the collection device, improper mixing, or exposure to air.[1,2,6]

9.1.3 Analysis is delayed (> 15 minutes for samples held at room temperature or > 60 minutes for samples held at 4°C).[1,2,4,6-8]

ABS 10.0 RESOURCES:

10.1 Recommended equipment

10.1.1 Single puncture: Appropriate anticoagulant, sterile glass or plastic (low diffusibility) syringe with needle, patient label.[6] 70% isopropyl alcohol or other suitable antiseptic solution,[1,7,8] gauze squares or similar material, well-fitting latex or vinyl gloves, puncture-resistant container, syringe cap, 'cork' and device to remove needle from syringe.

10.1.1.1 The term cork describes any device designed to allow insertion of the sample needle point after withdrawal from the artery. The purpose of the cork is to reduce exposure time of the sampler to the contaminated needle and remind the sampler not to resheath the needle—thus reducing the potential for inadvertent needle stick. The cork should provide some resistance to insertion and should not allow the needle to completely traverse it. Devices that allow single-hand recapping are preferred.

10.1.1.2 Local anesthetic is not generally considered necessary for single punctures.

10.1.2 Indwelling catheter: Sterile glass or plastic (low diffusibility) syringe that has been appropriately anticoagulated, 'waste' syringe,[1,7,8] syringe cap, protective eyewear and outerwear (in the anticipation of splashing), well-fitting latex or vinyl gloves, and patient labels[6-8] (local anesthetic is recommended for arterial line insertion)

10.1.3 Container of ice and water (to immerse syringe barrel if specimen will not be analyzed within 15 min)

10.1.4 A detailed institutional protocol incorporating current OSHA (Occupational Health and Safety Administration) and CDC (Centers for Disease Control) guidelines should be in place

10.2 Personnel:

Arterial blood sampling should be performed under the direction of a physician specifically trained in laboratory medicine, pulmonary medicine, anesthesia, or critical care. Two levels of training and experience are recognized for the actual sampling.

In addition, as one aspect of a quality assurance monitoring program, individuals performing arterial puncture should undergo periodic re-evaluation relative to Universal Precautions, proper syringe preparation, site determination,

puncture technique, sample acquisition, storage and disposal of blood specimens, and post-sampling care of puncture site. Individuals who do not meet acceptable indicator thresholds should not continue to perform punctures independently until they have received remedial instruction and been re-evaluated. (Please note that failure to obtain a sample from a single arterial puncture does not indicate failure as long as all other aspects of the indicator are acceptable.)

10.2.1 Level I: Persons designated as Level I should have a high school education plus specific training in sampling arterial blood, oxygen delivery devices and related equipment, recordkeeping, and the associated hazards and sources of specimen and sampler contamination. Additionally, a strong background in mathematics, with one or more years of college courses in the physical and biologic sciences is preferred.[18] Performance of blood sampling should be supervised by a Level-II individual.[18]

10.2.2 Level II: The Level-II person is a health care professional trained in patient assessment, acid-base, and oxygenation disorders, and diagnostic and therapeutic alternatives—an associate or higher degree in the sciences or respiratory therapy or subtantial experience in pulmonary function technology is preferred. Two years of college with courses in the biologic sciences and mathematics plus 2 years of training and experience may be substituted for personnel supervising arterial blood sampling. Level-II personnel both sample and supervise Level-I personnel during sampling.[18] A recognized credential (MD, DO, CRTT, RRT, RN, RPFT, CPFT, MT, MLT, RCVT, or equivalent) is strongly recommended.

ABS 11.0 MONITORING:

The following should be monitored as part of arterial blood sampling:

11.1 F_{IO_2} (analyzed) or prescribed flowrate[1,2,7,8]
11.2 Proper application of patient device (eg, mask or cannula)[1,2,7,8]
11.3 Mode of supported ventilation and relevant ventilator settings[7,8]
11.4 Pulsatile blood return[1,6-8]
11.5 Presence or absence of air bubbles or clots in syringe or sample[1,2,6-8]
11.6 Patient's respiratory rate[7,8]
11.7 Patient's temperature[7,8]
11.8 Position and/or level of activity (if other than resting)
11.9 Patient's clinical appearance
11.10 Ease of (or difficulty with) blood sampling[1,7,8]
11.11 Appearance of puncture site after direct pressure has been applied and before application of pressure dressing for potential hematoma formation[1,6-8] (a detailed protocol for post-sampling management should be in place)

ABS 12.0 FREQUENCY:

The frequency with which sampling is repeated should depend on the clinical status of the patient and the indication for performing the procedure[2,5] and not on an arbitrarily designated time or frequency.

12.1 Repeated puncture of a single site increases the likelihood of hematoma, scarring, or laceration of the artery. Care should be exercised to use alternate sites for patients requiring multiple punctures.
12.2 An indwelling catheter may be indicated when multiple sampling is anticipated.

ABS 13.0 INFECTION CONTROL:

13.1 Universal Precautions as published by the Centers for Disease Control[19] and directives issued by the Department of Labor concerning occupational exposure to blood-borne pathogens[20] must be applied in all circumstances involving blood or blood-contaminated collection devices in the immediate area.
13.2 Aseptic technique must be employed whenever blood is sampled from an indwelling arterial catheter.[1]

13.3 Prior to a single puncture, the site should be cleaned.[1,7,8]

13.4 Blood specimens, contaminated needles, and syringes must be disposed of in appropriate containers.

13.5 Needle sticks are the most frequent source of transmission of blood-borne diseases in health care workers.[19,20]

13.5.1 Needles used for blood sampling should be resheathed only with a technique that utilizes a one-hand device or by careful insertion into a cork, rubber plug, or similar device that prevents the sharp point from being accessible.

13.5.2 The needle should removed from the syringe and the syringe capped.

13.5.3 Gloves provide little protection from needle punctures but should be worn to prevent splashing of blood on sores or other skin breaks.

13.5.4 Specimen sampling devices in which the needle retracts after use are recommended when the design does not interfere with obtaining the sample.

Cardipulmonary Diagnostics Guidelines Committee:

Kevin Shrake MA RRT, Chairman, Springfield IL
Susan Blonshine RPFT RRT, Lansing MI
Robert Brown BS RPFT RRT, Madison WI
Gregg Ruppel MA RPFT RRT, St Louis MO
Jack Wanger MBA RPFT RRT, Denver CO
Michael Kochansky CRTT RCPT RPFT, York PA

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AARC Clinical Practice Guideline

Oxygen Therapy in the Home or Extended Care Facility

OT-CC 1.0 PROCEDURE:

The procedure addressed is the administration of oxygen therapy in the home or in the extended care facility other than by mechanical ventilator.

OT-CC 2.0 DESCRIPTION/DEFINITION:

Oxygen therapy is the administration of oxygen at concentrations greater than that in ambient air with the intent of treating or preventing the symptoms and manifestations of hypoxia.[1]

OT-CC 3.0 SETTING:

This Guideline is confined to oxygen administration in the home or extended care facility.

OT-CC 4.0 INDICATIONS:

Documented hypoxemia: In adults, children and infants older than 28 days: (1) $P_{aO_2} \leq 55$ torr or $S_{aO_2} \leq 85\%$ in subjects breathing room air.[1,2] or (2) P_{aO_2} of 56-59 torr or S_{aO_2} or $S_{pO_2} \leq 89\%$ in association with specific clinical conditions (eg, cor pulmonale, congestive heart failure, or erythrocythemia with hematocrit > 56).[3,4]

Some patients may not qualify for oxygen therapy at rest but will qualify for oxygen during ambulation, sleep, or exercise. Oxygen therapy is indicated during these specific activities when S_{aO_2} is demonstrated to fall to $\leq 88\%$.[5]

OT-CC 5.0 CONTRAINDICATIONS:

No absolute contraindications to oxygen therapy exist when indications are present.

OT-CC 6.0 PRECAUTIONS AND/OR POSSIBLE COMPLICATIONS:

6.1 In spontaneously breathing hypoxemic patients with chronic obstructive pulmonary disease, oxygen administration may lead to an increase in P_{aCO_2}.[6-8]

6.2 Undesirable results or events may result from noncompliance with physician's orders or inadequate instruction in home oxygen therapy.

6.3 Complications may result from use of nasal cannulae[9] or transtracheal catheters.[10]

6.4 Fire hazard is increased in the presence of increased oxygen concentrations.

6.5 Bacterial contamination associated with certain nebulizers and humidification systems is a possible hazard.[11-13]

6.6 Possible physical hazards can be posed by unsecured cylinders, ungrounded equipment, or mishandling of liquid oxygen (resulting in burns). Power or equipment failure can lead to an inadequate oxygen supply.

OT-CC 7.0 LIMITATIONS OF PROCEDURE:

Oxygen therapy has only limited benefit for the treatment of hypoxia due to anemia and benefit may be limited when circulatory disturbances are present.

OT-CC 8.0 ASSESSMENT OF NEED:

8.1 Initial assessment: Need is determined by the presence of clinical indicators as previously described and the presence of inadequate oxygen tension and/or saturation as demonstrated by the analysis of arterial blood. Concurrent pulse oximetry values must be documented and

reconciled with the results of the baseline blood gas analysis if future assessment is to involve pulse oximetry.

8.2 Ongoing evaluation or reassessment: Additional arterial blood gas analysis is indicated whenever there is a major change in clinical status that may be cardiopulmonary related. Arterial blood gas measurements should be repeated in 1-3 months when oxygen therapy is begun in the hospital in a clinically unstable patient to determine the need for long-term oxygen therapy (LTOT).[14] Once the need for LTOT has been documented, repeat arterial blood gas analysis or oxygen saturation measurements are unnecessary other than to follow the course of the disease, to assess changes in clinical status, or to facilitate changes in the oxygen prescription.[14,15]

OT-CC 9.0 ASSESSMENT OF OUTCOME:

Outcome is determined by clinical and physiologic assessment to establish adequacy of patient response to therapy.

OT-CC 10.0 RESOURCES:

10.1 Equipment

10.1.1 Low-flow oxygen systems: Such devices supply oxygen at flows that are less than the patient's inspiratory demand (ie, the delivered oxygen is diluted with room air). These devices may supply either a low or high F_{IO_2} depending upon the specific design.[16,17] Reservoir masks or other devices designed to provide for a high F_{IO_2} are usually not appropriate for long-term oxygen therapy outside of the hospital.

10.1.1.1 Nasal cannulae can provide 24-40% oxygen (depending on patient's inspiratory flowrate) with flowrates up to 6 L/min in adults;[1] infant flows should be limited to a maximum of 2 L/min.[18,19] Oxygen supplied to adults by nasal cannulae at flows ≤ to 4 L/min need not be humidified.[20,21]

10.1.1.2 Transtracheal oxygen catheters can provide continuous oxygen therapy with oxygen consumption compared to nasal cannulas of only about one half of that used at rest and two thirds of that used with exercise. Transtracheal catheters may require greater patient supervision and have an increased risk for complication.[10]

10.1.1.3 Oxygen-conserving devices: Oxygen reservoir cannulae (nasal or pendant), demand oxygen delivery devices, and transtracheal catheters are currently being utilized for oxygen conservation. Each of these devices may have advantages or disadvantages that are related to specific design.

10.1.2 High-flow oxygen delivery systems: Such devices can provide a prescribed gas mixture of high or low oxygen concentration at flows that exceed patient demand; however, they are impractical for home use except for humidifying devices that are compressor-driven with supplemental oxygen bled in at low flows. Tracheostomy collars and T-tube adapters may be used with high-flow supplemental oxygen systems. The gas should be humidified by a continuous aerosol generator or a heated humidifier.[22,23] The humidifier is preferable because of the greater likelihood for the transmission of contagion via nebulizer.

10.1.3 Oxygen supply systems

10.1.3.1 Oxygen concentrators: Concentrators extract oxygen from ambient air and should deliver oxygen at concentrations of 85% or greater at up to 4 L/min.[24] Membrane oxygen concentrators typically provide oxygen at a lower F_{DO_2}, and higher flows are required to 'compensate' for the reduced F_{DO_2}.

10.1.3.2 Liquid oxygen systems: Liquid oxygen is provided in large reservoir cannisters with smaller portable units that can be transfilled by the patient. There is evaporation loss from the cannisters when they are not in use.

Many of the portable liquid oxygen units are appropriate for ambulatory therapy and can be utilized with oxygen-conserving devices to extend their functional time.

10.1.3.3 Compressed gas cylinders: Oxygen may be supplied in large cylinders (eg, G or H cylinders) as stationary units for home oxygen therapy. Smaller cylinders (eg, D and E cylinders) may be equipped with wheels and used as portable oxygen units (strollers). E cylinders may also be used as a backup for oxygen concentrators in the event of a power or equipment failure. Smaller cylinders coupled with oxygen conserving devices may be used as ambulatory units.

10.2 Personnel: Credentialed respiratory care practitioners (RRT or CRTT) or other credentialed persons with equivalent training and documented ability to perform the tasks may assess patients, initiate and monitor oxygen delivery systems, recommend changes in therapy, and instruct caregivers.

Caregivers may operate and maintain oxygen delivery devices and assess a specific patient after they have been instructed by credentialed practitioners and have demonstrated the appropriate level of skill.

OT-CC 11.0 MONITORING:

11.1 Patient

11.1.1 Clinical assessment should routinely be performed by the patient and/or the caregiver to determine changes in clinical status (eg, use of dyspnea scales and diary cards). Patients should be visited/monitored at least once a month by credentialed personnel unless conditions warrant more frequent visits.

11.1.2 Measurement of baseline oxygen tension and saturation is essential before oxygen therapy is begun.[5,15] These measurements should be repeated when clinically indicated or to follow the course of the disease. Measurements of S_{O_2} also may be made to determine appropriate oxygen flow for ambulation, exercise, or sleep.

11.2 Equipment Maintenance and Supervision: All oxygen delivery equipment should be checked at least once daily by the patient or caregiver. Facets to be assessed include proper function of the equipment, prescribed flowrates, F_{DO_2}, remaining liquid or compressed gas content, and backup supply. A respiratory care practitioner or equivalent should during monthly visits reinforce appropriate practices and performance by the patient and caregivers and assure that the oxygen equipment is being maintained in accordance with manufacturers' recommendations. Liquid systems need to be checked to assure adequate delivery.[25] Oxygen concentrators should be checked regularly to assure that they are delivering 85% oxygen or greater at 4 L/min.[24]

OT-CC 12.0 FREQUENCY:

Oxygen therapy should be administered continuously unless the need has been shown to be associated only with specific situations (eg, exercise and sleep).

OT-CC 13.0 INFECTION CONTROL:

Under normal circumstances low-flow oxygen systems without humidifiers do not present a clinically important risk of infection and need not be routinely replaced. High-flow systems that employ heated humidifiers or aerosol generators, particularly when applied to subjects with artificial airways, can be important sources of infection and should be cleaned and disinfected on a regular basis. (Although studies have suggested that tubing should be changed every 48 hours in the hospital, there are no definitive studies regarding the frequency of tube changes at home or in long-term care facilities.)

Oxygen Therapy Guidelines Committee:

Dianne Lewis MS RRT, Chairman, Naples FL
Thomas A Barnes EdD RRT, Boston MA
Kay Beattie BA RRT, Columbus OH
Laura J Reisman Beytas MPH RRT, Peoria IL
Walter J O'Donohue Jr MD, Omaha NE
Noah Perlman BS RRT, Boston MA
Ray H Ritz BA RRT, Boston MA
John Salyer RRT, Salt Lake City UT

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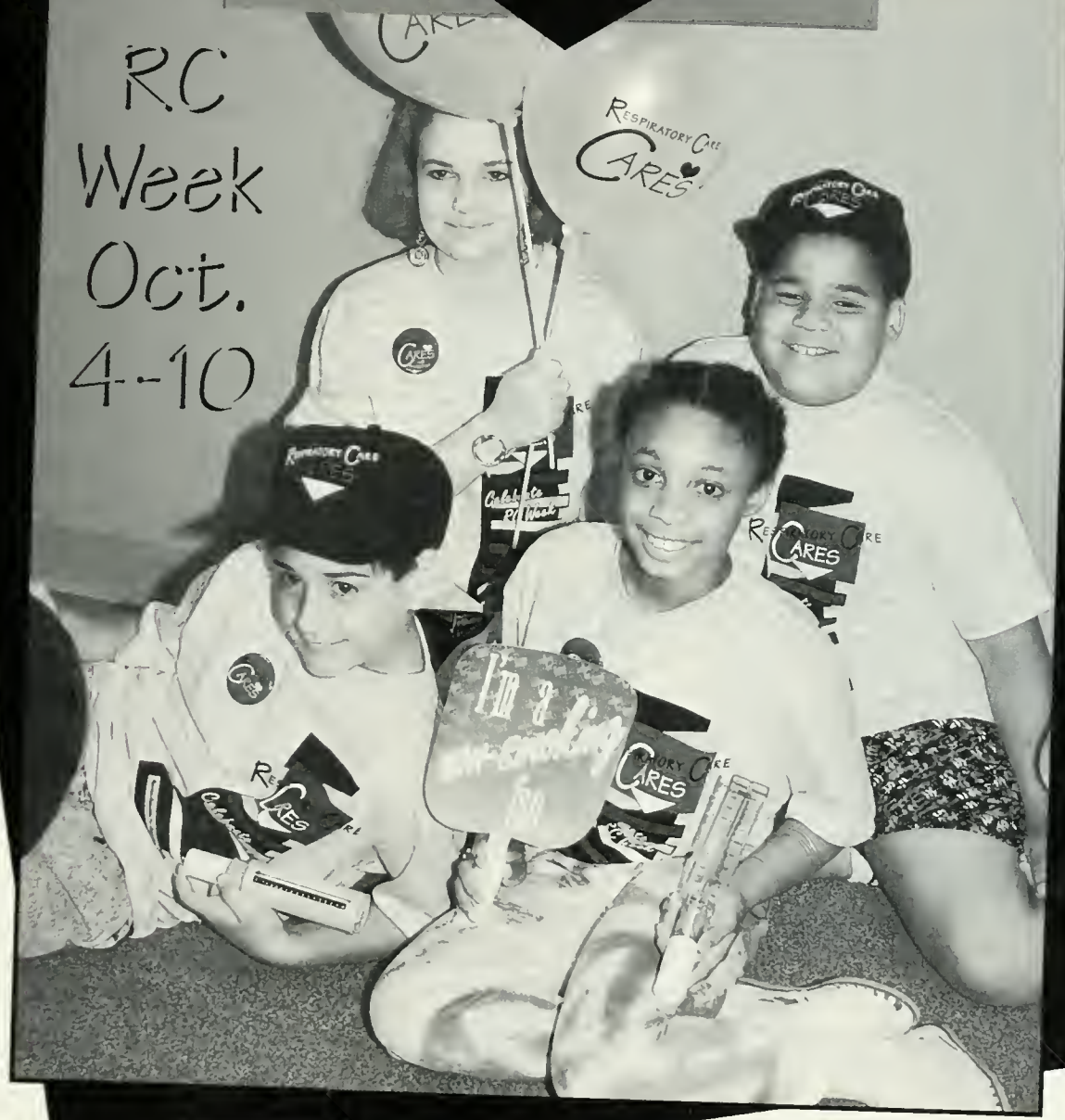

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Blood Gas Corner #30— Lethal Infantile Mitochondrial Disease

Mary P Martinasek RRT and Dushan J Martinasek MD

A 2-day-old term infant with an unremarkable prenatal and birth history developed lethargy, duskiness, and poor feedings. Physical exam was remarkable for an agitated, hypotonic, dusky infant in apparent respiratory distress. Vital signs were normal and bilateral breath sounds were clear. Cardiovascular examination was remarkable only for a markedly delayed capillary refill time. Chest x-ray suggested an enlarged heart, but vasculature appeared to be normal and no infiltrates were evident. Pulse oximetry revealed oxygen saturation measurements in the low- to mid-80s in room air. The infant was placed in a 100% oxygen head hood, and subsequent oxygen saturation readings were in the mid-90s. Initial capillary blood gas results with the infant in a 100% oxygen head hood were pH 6.72, P_{cCO_2} 53 torr [7 kPa], P_{cO_2} 65 torr [8.6 kPa] with an unrecordable base deficit. The infant was intubated, and assisted ventilation was begun. Plasmanate (Miles Inc, Elkhart IN) was administered for poor perfusion. Sodium bicarbonate was given for the profound acidosis, and prostaglandin therapy to prevent closure of the ductus arteriosus was initiated, in view of the long transport distance to the Level III nursery. The capillary blood gas values with the infant on 100% O_2 just prior to transport were pH 6.94, P_{cCO_2} 25 torr [3.3 kPa], P_{cO_2} 157 torr [20.9 kPa], and base deficit –28 mEq/L. More bicarbonate was gradually administered during the transport to the tertiary care center. Over the next few hours, with the infant mechanically ventilated and sedated, the blood gas values improved.

Admission laboratory results at the tertiary care center revealed an increased white blood cell count with a normal differential. The urine was negative for group B streptococcal antigen. The resting serum lactate concentration was 211 mmol/L (normal units < 20 mmol/L) and the pyruvate 4.75 mmol/L (normal units < 1 mmol/L).

Study Questions

1. How do you interpret the results of the blood gas analysis?
2. What should be considered in the differential diagnosis?
3. What is the significance of the elevated resting serum lactate and pyruvate concentrations?

Answers and Discussions on Next Page

Ms Martinasek is associated with the Transport Team, Children's Hospital Medical Center, and Dr Martinasek is a Fellow, Division of Neonatology, Department of Pediatrics, University of Cincinnati Medical Center—Cincinnati, Ohio.

Answers

1. Interpretation. The first blood gas sample demonstrates a severe metabolic acidosis combined with a respiratory acidosis. The second sample, after the administration of bicarbonate therapy and the initiation of mechanical ventilation, continues to demonstrate a metabolic acidosis that is partially compensated for by a respiratory alkalosis. The interpretation of capillary blood gas samples in the newborn should be done with caution. Some studies have shown a good correlation between arterial and capillary pH and P_{CO_2} values, but a poor correlation between the P_{O_2} values.[1-3] Others have found that capillary samples poorly predict arterial values of pH, P_{CO_2}, and P_{O_2}.[4] The P_{CO_2} values of 65 torr and 157 torr indicate adequate oxygenation for this infant as capillary values do not overestimate oxygenation.[2-4] Arterial samples later confirmed the extent of this infant's acidosis and ruled out the possibility of inaccurate capillary samples based on a poorly perfused sample site.

2. Metabolic Acidosis. In the term newborn, persistent metabolic acidosis is most commonly caused by hypoxia from cardiopulmonary disease, sepsis, congenital heart disease, or from an inborn error of metabolism. With adequate oxygenation as exhibited by the blood gases and with the chest radiograph free of infiltrates, the acidosis was determined not to be caused by hypoxia from cardiopulmonary disease.

Sepsis should be considered in the differential diagnosis of a metabolic acidosis in the newborn. However, the normal pulmonary component of the chest radiograph, the negative urine agglutination test, and subsequent normal blood counts with a negative blood culture in this normotensive newborn combined to make sepsis an unlikely etiology for the profound acidosis.

A severe metabolic acidosis may develop in an infant whose heart lesion precludes adequate circulation when the ductus arteriosus closes off (typically at a few days of life) and the region distal to the ductus becomes ischemic. Congenital heart disease was considered and prostaglandin therapy to provide ductal patency was initiated and maintained until a structural heart lesion had been ruled out by echocardiogram.

The presentation of sick newborns with an inborn error of metabolism is nonspecific. The response usually manifests itself in abnormal cardiorespiratory, gastrointestinal, and neurologic functions. Nonspecific symptoms may include poor perfusion, tachypnea, grunting, poor feeding, emesis, lethargy, jitteriness, or seizures. Although symptoms may progress rapidly, they usually present insidiously becoming more severe with time. Many patients with an inborn error of metabolism are not diagnosed for months or years. The probability of an inborn error of metabolism as the cause of the severe acidosis in this infant was increased as cardiopulmonary disease, sepsis, and congenital heart disease were eliminated from the differential.

3. Lactate and Pyruvate. Pyruvate is produced anaerobically from glucose via glycolysis with the production of a limited number of energy-rich adenosine triphosphate (ATP) molecules. The most efficient production of ATP requires aerobic conditions within mitochondria where pyruvate is a precursor in fueling the Krebs cycle. When the Krebs cycle is overwhelmed, such as in strenuous exercise, pyruvate is instead converted to lactate with the production of minimal ATP. Elevated lactate and pyruvate concentrations reflect a metabolic acidosis with this infant's inability to effectively utilize the Krebs cycle.

Discussion

Lethal infantile mitochondrial disease (LIMD) is a very rare disorder with only 19 prior cases having been reported. A defect in the mitochondria, the "power plants of the cell," results in impaired production of ATP, the high-energy molecule used for the majority of the body's activities. Oxidative phosphorylation is the process by which ATP is generated as electrons are passed along groups of respiratory complexes in the inner mitochondrial membrane. These respiratory complexes are collectively referred to as the electron transport chain. Electrons are provided for the electron transport chain from by-products of glucose metabolism to pyruvate in the cell cytosol and from products of the Krebs cycle in the cell mitochondria. In the production of ATP, electrons are transferred in complex I and II to coenzyme Q, then on to complex III (cytochromes b and c_1), to cytochrome c, to IV (cytochrome aa_3), and finally to oxygen, the terminal electron acceptor.[5]

Clinical manifestations of disorders of mitochondrial function range from mild muscle ache and weakness after prolonged exercise to severe or fatal lactic acidosis that develops within a few days of birth.[6] Patients with LIMD appear normal at birth but the ability of the mitochondria to carry out oxidative phosphorylation rapidly declines due to a defect involving complex I, complex IV, and complex III in the electron transport chain.[7] When infants with LIMD experience any type of increased activity, lactate concentrations increase in the blood as the dysfunctional mitochondria fail to generate sufficient ATP and pyruvate is instead converted to lactate. This patient became acidotic with activities such as suctioning, intravenous-line insertions, stimulation, and crying.

A high index of suspicion is necessary to diagnose mitochondrial disease or an inborn error of metabolism in the acidotic infant. Any hypotonic infant with lactic acidosis whose symptoms cannot be explained by asphyxia, sepsis, or cardiopulmonary disease should be evaluated for an inborn error of metabolism or a mitochondrial defect. The reader is referred to recent reviews to aid in the diagnostic approach.[5,7-9]

The treatment of infants with LIMD is with vitamin C, vitamin K, and coenzyme Q.[7] This form of combined electron-acceptor therapy attempts to assist the flow of electrons down the electron transport chain, thereby increasing mitochondrial ATP production and halting the progression of the clinical manifestations. Coenzyme Q assists in the transfer of electrons in the electron transport chain.[7] Vitamin K in conjunction with vitamin C donates electrons directly to cytochrome c which is needed for electrons to move from complex III to complex IV.[7]

Even with combined electron-acceptor therapy, the prognosis in patients with LIMD is poor, with death typically occurring within the first year of life.[7]

Once combined electron-acceptor therapy was initiated, the presented infant responded well and was gradually weaned from the ventilator and from the heavy sedation that had been necessary to prevent him from developing overwhelming metabolic acidosis. He was discharged home at 3 months of age, but within 24 hours he had decompensated. Despite combined electron-acceptor therapy, continuous mechanical ventilation, heavy sedation, pharmacologic alkalinizing agents, and intermittent paralysis, he continued to deteriorate over the next 2 months. He eventually developed severe myocardial hypertrophy and congestive heart failure from which he was unable to recover. He died at 5 months of age.

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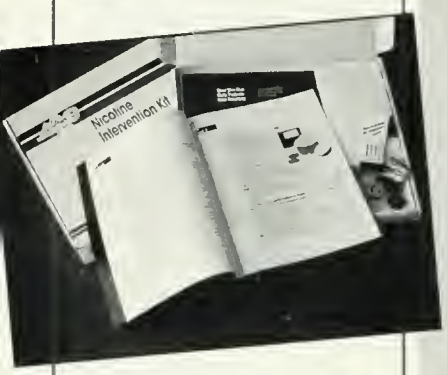

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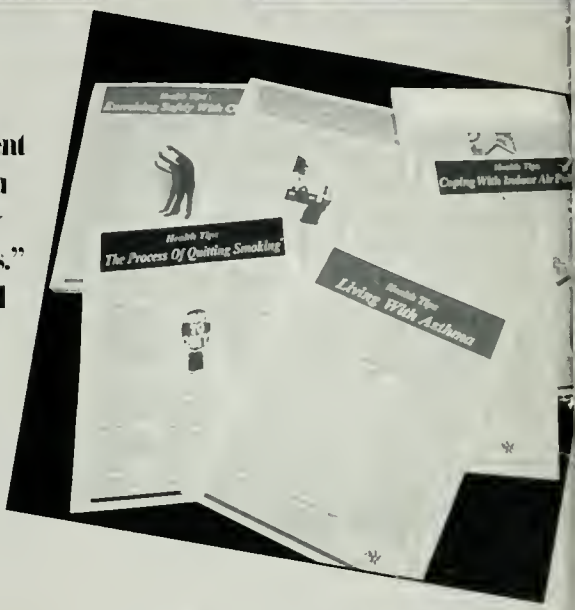

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PFT Corner # 44— Problems with a Bronchial Challenge

Jack Wanger MBA RPFT RRT and Charles G Irvin PhD

A 26-year-old woman was seen in a hospital's outpatient clinic for evaluation of her asthmatic symptoms and a second opinion. At that time, she was taking inhaled beta agonists, inhaled steroids, and inhaled ipratropium bromide. Physical exam revealed normal breath sounds and breathing pattern. Her height was 62 in. and she weighed 206 lb. The results of her initial pulmonary function tests are shown in Table 1 and Figure 1.

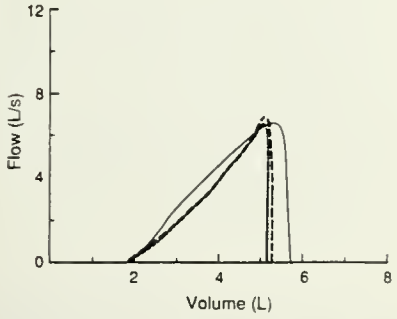

Fig. 1. The flow-volume curves for a 26-year-old woman with symptoms of asthma. Reference curve (predicted) = ——; before albuterol curve = – – –; after albuterol curve = ...

Table 1. Pulmonary Function Test Results before and after Bronchodilator (Albuterol) in a 26-Year-Old Woman Reporting Symptoms of Asthma

	Before	After
TLC (L)	4.23 (89)*	4.29
TGV(L)	1.84 (72)	1.98
RV (L)	0.86 (62)	0.94
FVC (L)	3.25 (89)	3.26
FEV_1 (L)	2.84 (87)	2.87
FEV_1/FVC	88%	88%
$FEF_{25-75\%}$ (L/s)	4.79 (121)	4.95
R_{aw} (cm $H_2O \cdot s \cdot L^{-1}$)	1.11	1.36
sG_{aw} ($s^{-1} \cdot cm\ H_2O^{-1}$)	0.30	0.30
D_{LCO} ($mL \cdot torr \cdot min^{-1}$)	27.2 (96)	
D_{LCO}/V_A	6.8 (115)	

*Values in parentheses are percent of predicted.

Questions

1. How do you interpret the pulmonary function test results?

2. What further tests do you recommend?

3. How do you interpret the results of the second diagnostic test (Table 2, Page 000)?

4. What problems do you observe with the methacholine challenge and what steps are indicated?

Answers and Discussion on Next Page

Mr Wanger is Manager and Dr Irvin is Associate Director of the Pulmonary Physiology Unit, National Jewish Center for Immunology and Respiratory Medicine, Denver, Colorado.

Answers

1. PFT Interpretation: The test results (Table 1 and Fig. 1) show a reduced thoracic gas volume (TGV) and residual volume (RV), with a normal total lung capacity (TLC). These changes are probably secondary to the patient's being overweight. The flow-volume loop is within normal limits, with no significant response to inhaled albuterol. The diffusing capacity (D_{LCO}) is normal.

2. Further Tests: With these results one might ask, Does this patient really have asthma? One way to answer this question is to perform a bronchial provocation test to determine whether airway hyperresponsiveness is present. The most common type of bronchial provocation test (sometimes called a challenge) involves the inhalation of progressively higher doses of aerosolized methacholine. Other challenges include the inhalation of aerosolized histamine, inhalation of cold air, and exercise.

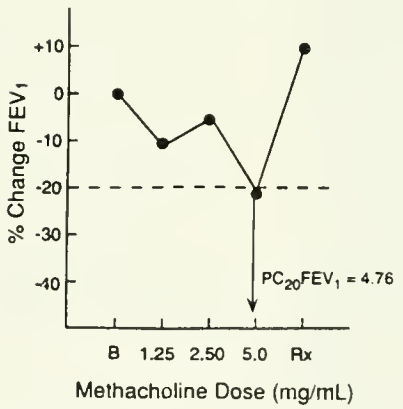

Fig. 2. A graphic representation of the first methacholine challenge. The % change in FEV_1 is calculated from baseline (B). The dosing begins at 1.25 mg/mL and ends at 5.0 mg/mL. The PC_{20} FEV_1 (provocative concentration that caused a 20% fall in FEV_1) is 4.76 mg/mL.

Table 2. Results of Bronchial Provocation Test with Inhaled Methacholine in a 26-Year-Old Woman To Determine Whether Airways Hyperresponsiveness Exists*

Methacholine Dose (mg/mL)	FEV_1	% Change†
Baseline	2.20	
1.25	1.96	–11
2.50	2.10	–5
5.00	1.74	–21
Albuterol administered	2.43	

* Technician noted that patient had used bronchodilator and steroid MDIs 2 hours prior to test and was coughing, had a runny nose, and was complaining of having the flu.
† Change from baseline.

3. Results of Second Diagnostic Test: The results of the second diagnostic test, a methacholine challenge, are shown in Table 2 and Figure 2. The results suggest that 5.0 mg/mL of methacholine is the PC_{20} FEV_1 (the methacholine concentration that provokes a greater than 20% fall in FEV_1); however, because of certain details related to the patient and the testing procedure, the results may be invalid.

4. Problems with the Methacholine Challenge: The technician noted on the report that the patient was coughing, had a runny nose, and complained of "having the flu" and had inhaled a beta agonist and steroids less than 2 hours prior to the test. Further, no diluent aerosol had been administered prior to administration of the provocative agent.

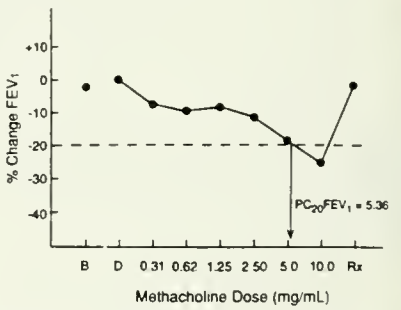

Fig. 3. A graphic representation of the second methacholine challenge. The % change in FEV_1 is calculated from the FEV_1 measured after inhalation of the diluent (D). The dosing begins at 0.31 mg/mL and increases in doubling increments up to 10 mg/mL. A bronchodilator is then administered after the 10 mg/mL dose because of a greater than 20% fall in FEV_1, and pulmonary function tests are repeated (Rx). The PC_{20} FEV_1 is 5.36 mg/mL.

Follow-Up and Discussion

Some weeks later a well-prepared patient free of the effects of illness was challenged for a second time, and the results are shown in Figure 3 and Table 3.

The methacholine challenge is an easy, safe, and reproducible test when a standardized protocol (eg, Chai et al[1]) is followed. Although this type of challenge is easy to perform, a number of factors may introduce variability and make interpretation difficult. These include patient preparation, formulation and storage of challenge materials, pulmonary function tests, aerosol delivery methods, and dosing.

Patient Preparation

Bronchial challenges require special attention to patient preparation. If the patient is not prepared properly or is not feeling well, postponement should be considered. The more important factors to consider in patient

Table 3. Results of a Second Bronchial Provocation Test with Inhaled Methacholine To Confirm Hyperresponsive Airways in a 26-Year-Old Woman

Methacholine Dose (mg/mL)	FEV_1	% Change*
Baseline	3.09	
Diluent only†	3.15	< 2
0.31	2.89	−8
0.62	2.85	−10
1.25	2.87	−9
2.50	2.78	−12
5.00	2.54	−19
10.00	2.34	−26
Albuterol administered	3.05	

*Percent change from diluent alone.
†0.9% saline.

preparation are the power of suggestion, the need to withhold medications that affect pulmonary function, and the presence or absence of viral infections.

Both bronchoconstriction and bronchodilation can be induced by suggestion.[2-4] The patient should be told as little as possible, but can be told that he or she will inhale a mist that will make breathing better, worse, or cause no change. Although suggestion may not have been an issue with our patient, it can be a problem.

Ideally, the patient should forego all medications prior to a bronchial challenge test. However, in many patients this is not possible. The American Thoracic Society (ATS)[5] and the European Society for Clinical Respiratory Physiology (SECPR)[6] suggest the following schedule for withholding pulmonary medications prior to a bronchial provocation test:

Inhaled beta adrenergics	4-12 hours
Anticholinergic aerosols	12 hours
Cromolyn sodium	8 hours
Oral beta adrenergics	12 hours
Theophyllines	24-48 hours
H_1-receptor agonists	48 hours

Prior to the first methacholine challenge (Table 2), the patient used inhaled beta agonists and inhaled steroids less than 2 hours before the test. Knowing this, the technician should have notified the ordering physician to discuss postponement. These treatments would have lead to an underestimation of the hyperresponsiveness present.

Because the patient probably had a viral infection, took medications near the start of the test, and did not inhale a diluent prior to inhaling the methacholine, the results are difficult to interpret and may be invalid. The challenge should be repeated when the patient has fully recovered from the effects of the viral infection (this may take as long as 6 weeks).[7] Particular care should be taken to instruct the patient to forego medications that affect pulmonary function for an adequate period before the test, and, finally, the patient should be challenged with aerosolized diluent before aerosolized methacholine is administered.

Viral infections can result in increased hyperreactivity.[8] Our patient was symptomatic and claimed to have an influenza-like illness. Again, knowing this, the technician should have notified the ordering physician to discuss postponement. This factor unlike the medications would have caused an overestimation of the hyperresponsiveness. Performing the test under the described conditions makes the results difficult to interpret.

Standardized Methacholine Challenge

The methacholine chloride crystals are dissolved in saline (also called diluent). When beginning a methacholine challenge test, the patient should first inhale the diluent without methacholine from the same nebulizer-delivery system and in the same manner that the diluted methacholine is to be inhaled. The reasons for this are (1) to determine whether the patient reacts to the inhalation of the aerosolized diluent, and (2) to establish the control from which percent-change will be calculated. Pulmonary function tests should be repeated 1-3 minutes after inhalation of the diluent. If the fall in spirometry values is less than 10%, the challenge can proceed.

Pulmonary Function Tests

Baseline measurements of pulmonary function are usually made prior to a bronchial challenge to determine if contraindications exist. One commonly used guide to proceeding with the challenge is an $FEV_1 > 80\%$ of the patient's best recorded value.[3] In our patient, the baseline FEV_1 prior to the challenge (Table 2) was 2.20 L, which is approximately 77% of the FEV_1 value obtained with the initial tests (Table 1). The decrease in FEV_1 could have been caused by the influenza because the FEV_1 rose to 2.09 at the time of the second challenge.

Aerosol Delivery Methods

Many factors affect the amount of challenge material reaching the airway, including nebulizer output, par-

ticle size, amount of tubing between nebulizer and mouth, inspiratory flow-rate, breathing pattern, and breath-hold time. Nebulizer output is probably the most important of these. Therefore, care should be given to selection of the brand and model of nebulizer used. A study of the reproducibility of nebulizer output has shown an intrabrand coefficient of variation as high as 24% for a number of nebulizer brands and an intra-nebulizer coefficient of variation as high as 10%.[9] Thus, the same nebulizer should be used for each dose and for each repeated test in the same patient. In some laboratories, the various doses of methacholine are set up in different nebulizers prior to beginning the test. If this practice is followed, output from each nebulizer should be routinely measured to ensure similar outputs.

Dosing

The starting dose for a methacholine challenge should be low (eg, 0.03 to 0.15 mg/mL) when patients suspected of being hyperresponsive are being tested. After the initial dose, concentrations should be increased in doubling increments until the test is positive (a fall in FEV_1 of $\geq 20\%$ is observed) or the highest dose is reached. Methods that employ higher initial doses have been described,[10-12] but these should be used with caution.

In Conclusion

The use of bronchial provocation tests has increased because they are safe and useful tools for the evaluation of airway hyperreactivity. However, one must be alert to such issues as patient preparation, contraindications, and techniques that can result in false-negatives, false-positives, or uninterpretable data.

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How Ventilators Provide Temporary O_2 Enrichment: What Happens When You Press the 100% Suction Button?

Robert S Campbell RRT and Richard D Branson RRT

Background

100% O_2 Suction (definition): *A ventilator control to provide 100% oxygen, primarily for hyperoxygenation immediately before and after endotracheal suctioning.*

Patients requiring artificial airways for airway protection and/or mechanical ventilation must be suctioned with some frequency for adequate removal of pulmonary secretions. The endotracheal suctioning procedure is not without complication, however, as evidenced by the list of reported adverse reactions (Table 1).[1-21]

Table 1. Reported Complications of Endotracheal Suctioning

Cardiac arrest[3]
Cardiac arrhythmias[4]
Hypoxemia[1-14]
Mucosal trauma[15,16]
Atelectasis[6,9,17]
Increases in intracranial pressure (in infants)[18,19]
Infection[20,21]

Hypoxemia is certainly the most frequent complication of endotracheal suctioning and can be implicated as inciting some of the other more serious, life-threatening complications of the procedure. The etiology of suction-induced hypoxemia is multifactorial and usually includes one or several of the following: (1) evacuation of gas from the lungs, (2) entrainment of room air into the lungs, (3) interruption of mechanical ventilation, (4) loss of PEEP/CPAP, (5) amount of negative pressure applied, (6) ratio of the diameter of suction catheter to diameter of endotracheal tube, (7) duration of suction procedure, and (8) ventilator variables. Many studies have been devoted to preventing and minimizing the severity and duration of arterial desaturation caused by endotracheal suctioning. These studies attempt to reduce suction-induced hypoxemia either by optimizing the procedural technique used (ie, hyperoxygenation, hyperinflation, hyperventilation, duration of suctioning, negative pressure used, ratio of suction catheter diameter to endotracheal tube diameter) or by the use of an additional device (ie, closed-suction system,[11,22] double-lumen suction catheter for oxygen insufflation during suctioning,[8,10,14,23] swivel adapter,[24,25] jet ventilator[26,27]). A universal finding among these studies is that pre- and postoxygenation reduces suction-induced hypoxemia. No single technique is currently accepted as a standard for pre- and postoxygenation of mechanically ventilated patients requiring endotracheal suctioning. In fact, these techniques may vary widely among institutions or even among ICUs in the same institution! Brown et al[11] studied the advantages and disadvantages of closed- and open-system suctioning techniques and concluded that the closed system is preferable.

We believe that it is important for practitioners who utilize the closed-suction system to be aware of the disadvantages of this technique. First, the mechanical ventilator cannot be as responsive to the patient's changing ventilatory needs or drive during the suctioning procedure as can manual ventilation in the hands of a trained respiratory care practitioner. Manual ventilation with a self-inflating bag may also improve the mobilization of pulmonary secretions and improve the practitioner's 'feel' for the patient's secretions, compliance, and resistance. Second, practitioners should be aware of the effects of ventilator mode when using the closed-suction system. Craig et al[22] have shown that significant desaturation can occur in patients in the assist/control mode due to the

flow deficit in a closed-airway system. Third, in 1979, Benson and Pierson[28] described how a ventilator's washout volume creates a lag time when the delivered oxygen concentration (F_{DO_2}), and thus the F_{IO_2}, is altered. The washout volume is the internal volume of the mechanical ventilator that must be flushed out before an increase in F_{DO_2} is seen at the inspiratory outlet. The lag time is affected directly by the ventilator washout volume and the patient's minute volume. All of the ventilators that have a 100% O_2 Suction feature utilize microprocessor-controlled proportional valves to set F_{IO_2} and most do not possess internal reservoirs. Because the washout volume varies by manufacturer, it is listed for each ventilator. Currently, five ventilator manufacturers have accommodated practitioners with the addition of a 100% O_2 Suction feature to aid and simplify the pre- and post-oxygenation of patients requiring endotracheal suctioning. The ventilators that utilize a 100% O_2 Suction program are listed, and program characteristics are shown in Table 2.

100% Oxygen Features

BEAR 5

The 100% O_2 feature on the BEAR 5 (Bear Medical Systems Inc, Riverside CA) is activated by pressing the 100% O_2 key and the Enter key in sequence. The delivery of 100% O_2 is terminated automatically after 3 minutes or can be manually terminated by pressing the 100% O_2 and Enter keys in sequence a second time. Termination of the program causes the ventilator to revert to the original F_{IO_2} setting. The red indicator light on the 100% O_2 key is illuminated to alert the user that it is activated. All alarms remain active at the user-selected settings during the 3-minute period. The washout volume of the BEAR 5 is 3.6 L due to an internal reservoir.

Puritan-Bennett 7200ae

The 7200ae (Puritan-Bennett Corp, Carlsbad CA) has a 100% O_2 Suction feature that is activated by pressing the 100% O_2 Suction key and the Enter key in sequence. The delivery of 100% O_2 is terminated automatically after 2 minutes or can be manually terminated by pressing the 100% O_2 Suction and Enter keys in sequence a second time. This causes the ventilator to revert to the original F_{IO_2} setting. The 7200ae alerts the user that the feature is operational by (1) illuminating the red indicator light on the 100% O_2 Suction key, (2) flashing 100 in the O_2 window display in the settings section of the control panel, and (3) displaying the status of the 100% O_2 Suction feature (on/off) on the Ventilator Settings 2 screen (if the ventilator is equipped with the 7202 display screen). The O_2 value

Table 2. Characteristics of 100% O_2 Suction Features among Adult Mechanical Ventilators

Ventilator	Activation	Duration (until automatic termination)	Manual Termination	Alarms	Washout Volume
BEAR 5	Touch key entry	3 min	Yes	Active	3.6 L
Infrasonics AdultStar	Rotary knob, touch key entry	3 min	Yes	Active	200 mL
PPG IRISA	Touch key × 3 s	Preoxygenation 3 min; suction time 2 min; postoxygenation 2 min	Yes	Audible, suspended	40 mL
P-B 7200ae	Touch key entry	2 min	Yes	Active	60 mL
Servo 300	Twist knob	20 breaths or 1 min, whichever occurs first	Yes	Active	50 mL

displayed on the Ventilator Settings 1 screen continuously shows the user-selected F_{IO_2} level. All alarms remain active at the user-selected settings during the 2-minute period. Washout volume of the 7200ae ventilator is approximately 60 mL.

Infrasonics AdultStar

The 100% O_2 for 3 Min feature of the AdultStar (Infrasonics Inc, San Diego CA) is accessed from the Ventilator Settings screen. A backlit cursor is moved to the F_{IO_2} setting by rotating the control knob. Once the F_{IO_2} setting is highlighted with the cursor, the Enter button is depressed to enable the rotary knob to change the F_{IO_2} value. By rotating the knob clockwise beyond the 100% setting, the 100% O_2 for 3 Min feature is displayed and is activated by depressing the Enter key. The AdultStar delivers 100% O_2 for 3 minutes and alerts the user by displaying 100% O_2 for 3 Min with a flashing asterisk (*) on the Ventilator Settings screen. The 100% O_2 for 3 Min feature can be manually terminated by using the control knob to return the F_{IO_2} to the previous setting and pressing the Enter button. All alarms remain active at user-selected settings during the 3-minute period. The washout volume of the AdultStar is approximately 200 mL.

Servo 300

The Servo 300 ventilator (Siemens Medical Systems Inc, Patient Care Systems Div, Piscataway NJ) has an O_2 Breath feature that is part of a dual-function knob in the control section. This feature is activated by turning the knob toward O_2 Breath (clockwise). The F_{IO_2} automatically returns to the control setting following either the delivery of 20 breaths (machine + spontaneous) or after 1 minute has elapsed, whichever comes first. The feature may also be manually terminated by turning the O_2 Breath knob clockwise a second time. The 300 alerts users that the feature is operational by (1) illuminating a yellow indicator light next to the O_2 Breath knob, (2) immediately changing the digital readout for set F_{IO_2} to 100, and (3) gradually increasing the digital readout for measured F_{IO_2} as the washout volume is flushed out and the actual delivered F_{IO_2} increases to 100. The audible O_2 alarm is suspended during O_2 Breath operation. The washout volume of the Servo 300 is 50 mL.

PPG IRISA

The IRISA ventilator (PPG Biomedical Systems, Lenexa KS) utilizes a complex 100% O_2 Suction program. The program is initiated by depressing the 100% O_2 Suction key in the control section for 3 seconds. The program is divided into three stages: (1) preoxygenation with 100% O_2 for up to 180 seconds, (2) the suctioning procedure for up to 120 seconds, and (3) postoxygenation with 100% oxygen for 120 seconds. During Stage 1, the ventilator operates at the control settings, except oxygen concentration is increased to 1.0 and a minimum level of 4 cm H_2O PEEP is added (if set PEEP is < 4 cm H_2O) in order to sense patient disconnection. The F_{IO_2} alarm is inactive during this stage. Stage 2 begins when the patient is disconnected from the ventilator (sensed as loss of PEEP). If the patient is not disconnected within the 180 seconds of Stage 1, the suction program is inactivated. During Stage 2, ventilator operation and all audible alarms are suspended; however, visual alarms are activated when alarm limits are violated. The IRISA delivers a bias flow of 20 L/min during this stage in order to sense patient reconnection, which initiates Stage 3 of the program. If the patient is disconnected for longer than 120 seconds, the suction program is inactivated, the audible alarm sounds, and normal ventilator operation resumes. Stage 3 is similar to Stage 1 as the ventilator functions normally with 100% O_2 administered for 120 seconds; however, PEEP is maintained at the set value, and all audible alarms remain silenced for the duration of Stage 3. The user is alerted to the status of the program in two ways: (1) a green indicator light on the 100% O_2 Suction key is illuminated and (2) a message on the display screen informs the user of the stage in which the program is operating and displays the time remaining in seconds. The user is informed when the O_2 program is inactivated with a message on the graphic display and flashing of the indicator light on the 100% O_2 Suction key for 5 seconds. The 100% O_2 Suction program may be manually terminated at any point by pressing the Reset/Confirm key in the digital display section. The washout volume of the IRISA is 40 mL.

Commentary

Although the technique used to perform endotracheal suctioning of mechanically ventilated patients remains largely an institutional choice, we believe that certain portions of the procedure should be universally accepted and performed. Pre- and postoxygenation of patients requiring endotracheal suctioning should, in our opinion, be mandatory. The exact technique used to accomplish pre- and postoxygenation should remain flexible in order to meet individual patient needs, appropriately use available equipment, and minimize cost, if possible.

Many ventilator manufacturers have incorporated a temporary O_2 enrichment feature to aid, encourage, and simplify the use of increased oxygen concentrations during the pre- and postsuctioning periods. These features may be used for both closed- and open-system suctioning techniques and may also be clinically indicated for other procedures (eg, hyperoxygenation for ICU procedures such as chest x-rays, baths, bed changes, percussion and postural drainage, and line insertion, all of which have been associated with temporary arterial oxygen desaturation). The considerable variability among ventilator manufacturers' O_2 enrichment programs leads to the questions, What characteristics of temporary O_2 enrichment programs should be mandatory and which of the optional characteristics are useful? The mandatory characteristics include quick and easy activation, a display message or light alerting the user that the feature is operational, and automatic termination of the feature, with the option to manually terminate the feature if desired. We believe that many optional characteristics are desirable and worthwhile. It may be beneficial for the user to select the duration of the O_2 enrichment period (eg, for patients requiring more time to recuperate from the suctioning procedure or during certain procedures such as feeding tube or Swan-Ganz catheter placement). Automatic disabling of backup or apnea ventilation may also be desirable because patients are commonly disconnected and may then be returned to the same ventilator settings following the procedure. A useful option for increased patient and caregiver safety would be for the ventilator to minimize flow through the breathing circuit during patient disconnection. When the patient is disconnected, most ventilators purge the circuit with flow in an attempt to maintain PEEP or to deliver a mandatory breath. This flow can easily aerosolize contaminated condensate in the circuit and spray it towards the patient and/or the caregiver—placing all at increased risk of infection. Certainly these microprocessor-controlled ventilators have the potential to immediately sense disconnection, silence the alarms, and decrease the flow if the O_2 feature is activated prior to disconnection. The IRISA has already incorporated some of these features.

In Conclusion

Some form of pre- and postsuctioning hyperoxygenation is useful in minimizing or eliminating suction-induced hypoxemia. The purpose of this Corner is to inform users of the O_2 enrichment feature of the five adult ventilators that offer such a feature. It is equally important for practitioners who use other mechanical ventilators to preoxygenate patients requiring suctioning and to understand how the differences in ventilator washout volume and flushing capability affect F_{IO_2} delivery.

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Details related to specific aspects of products described are based on information supplied by the manufacturer unless otherwise noted.

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Reprints: Richard D Branson RRT, Dept of Surgery, Univ of Cincinnati Medical Ctr, 231 Betheseda Ave, Cinncinnati OH 45267-0558.

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The Toxic Effect of Tobacco Vapor, with Report of Cases*

(A Report in JAMA 100 Years Ago)

Dr. W. Carroll Chapman, of Louisville, said that usually the presence of tobacco poison in the systems of tobacco workers is manifested during the first day or two by violent vomiting, retching, purging and often a state of collapse, after which the system may become inured to it. Occasionally we find one whose constitution, even by contact and time, although there is a certain amount of toleration, refuses to receive it kindly, and emaciation begins, attended sooner or later by such symptoms as the following case illustrates:

Case I.—Willie C., aged 10 years, was found suffering extreme pain in the abdominal region, with the intensity centering at the umbilicus. Temperature, under the tongue, 100°; pulse 108, small, wiry and irregular; respiration 20 to 22, but irregular—several short, shallow respirations followed by one deep and gasping. Tongue glairy, red appearance and pointed. Patient constipated for the last several days; abdomen flat or rather depressed; urine scanty and slightly colored; skin dry, as were the hands and feet, the latter being a little cold. When near the patient the odor of tobacco was so pronounced that the doctor made inquiries regarding it and learned that he worked in a tobacco steamery and further, that he had slight attacks of similar pains at several different times except of a milder form.

As to the nicotianin, indications point strongly to its being a cause. According to Landerer it occurs only in dried tobacco leaves, and has the odor of that plant; a point strongly in its favor, as that odor was so distinct in every case the author had seen. It would seem further, that the basic substances and fatty acids were causative agents, because authors have proven, by physiological experiments, that these cause contraction of the pupil, dyspnoea, abdominal pains, convulsions and death.

The author directs attention to two factors noticeable in all the cases, namely: the emaciation, and the time each one had followed the occupation, that is, from six weeks to three months. The three cases which he reports had not suffered from the vomiting and retching usually attendant upon young tobacco workers the first day or two. In the other, or milder cases, he neglected to enquire regarding that point.

The toxic effects of tobacco vapor and its treatment was a subject worthy of more consideration than the profession has accorded it in the past, and he hoped that the next few years, aided by diligent and careful investigation, would place the matter in a more intelligent light.

*The material on which this report is based originally appeared in JAMA, Oct 31, 1891;17:699. The version presented here is reprinted, with permission, from "JAMA 100 Years Ago," in JAMA—the Journal of the American Medical Association, Dec 11, 1991;266:3087, having been edited by Annette Flanagin RN MA, Assistant to the Editor of JAMA, and Micaela Sullivan-Fowler MS MA. Copyright 1991, American Medical Association.

Books, Films, Tapes, & Software

Reviewer's comments: It is coincidental that two teaching manuals for bronchoscopy are reviewed this month. Readers who want a direct comparison should note the following: Both manuals are small (less than 200 pages), introductory in content, and current, and contain excellent figures and illustrations on glossy coated paper. The Stradling manual is more general and aimed at diagnosis, while the Ovassapian manual is more specific and aimed at controlling the airway. Neither provides sufficient information to set up or run an endoscopy laboratory because more specific information on maintenance, safety, infection control, and supplies is required. However, it is hoped that both will be available in every endoscopy laboratory.

Diagnostic Bronchoscopy: A Teaching Manual, 6th edition, by Peter Stradling MD. Hardcover, 185 pages, illustrated. New York: Churchill-Livingstone Inc, 1991. $133.95.

For nearly a quarter of a century, physicians learning bronchoscopy have consulted this manual, which succinctly reviews the indications, fiberoptic and rigid techniques, normal anatomy (including variants), inflammatory changes, disorders causing airway distortion, tumors, techniques for collecting specimens, and miscellaneous airway conditions (eg, sarcoidosis, radiation changes). By intent, the information is current and preparatory, the text is brief and dogmatic, and there are no references. The main reason for reading (and rereading) this book is its 234 color photographs of *outstanding* clarity, achieved by taking photographs through the rigid scope (to avoid image degradation by fiberoptics) and printing the images on glossy paper. Each photograph is accompanied by an explanatory line drawing; the photographs were inverted for this edition to accommodate the common practice of performing fiberoptic bronchoscopy while facing the patient. An index helps the user locate the color plates quickly; another 20 figures clarify teaching points.

The author is the recently retired Director of the Chest Clinic and Senior Lecturer in Respiratory Diseases at the Royal Postgraduate Medical School (Hammersmith Hospital) in London. Readers are the beneficiaries of his experience.

This manual is mandatory for bronchoscopists-in-training. Nurses and respiratory care practitioners who assist with bronchoscopy will also want to consult this book as an introduction to the images they will see on color video monitors.

Fiberoptic Airway Endoscopy in Anesthesia and Critical Care, by Andranik Ovassapian MD. Hardcover, 172 pages, illustrated. New York: Raven Press, 1990. $105.00.

Conceptually, this manual is divided into 3 sections. The initial section describes principles of fiberoptic endoscopes (bronchoscopes, laryngoscopes, and nasopharyngolaryngoscopes, dos and don'ts); anatomy of the airway: radiology of the airway, neck, and thorax; and topical anesthesia. The second section reviews the use of fiberoptic endoscopes for tracheal intubation, bronchial intubation (eg, for ventilating each lung separately), and airway management in the intensive care unit (eg, for checking endotracheal tube position or replacing an orotracheal tube with a nasotracheal tube). The final section reviews the difficult airway, difficult intubation, 10 case studies, and a progressive training plan. The writing is concise, tables are used liberally, and the references are pertinent. The color photographs are a remarkable improvement over those in the seminal text by Ikeda (**Atlas of Flexible Fiber-scopy**, published by University Park Press, Baltimore MD, 1974), but are not quite as clear as those in the Stradling text (**Diagnostic Bronchoscope: A Teaching Manual**, 6th edition, published by Churchill-Livingstone, New York NY, 1991).

The author of this manual, which is intended to illustrate use of fiberoptic endoscopy to manage the airway, is Professor of Anesthesiology at Northwestern University Medical School and Chief of the Anesthesia Service at the Veterans Administration Lakeside Medical Center in Chicago. Several of his colleagues contributed to some chapters.

While this book is aimed at anesthesiologists, it will be useful for physicians, nurses, and respiratory care practitioners who are involved in airway management.

John W Shigeoka MD
Chief, Pulmonary Section
VA Medical Center
Salt Lake City, Utah

Respiratory Therapy Examination Review, by Galen G Heath RRT and John M Gallagher RRT. Soft-cover, 200 pages. New York: Medical Examination Publishing Company, 1991. $24.00.

Comprehensive respiratory therapy review books have become popular as an effective way for students

and graduates to prepare for professional credentialing examinations. **Respiratory Therapy Examination Review** is written primarily for students preparing to take the National Board for Respiratory Care (NBRC) entry-level exam and is designed to provide an opportunity to improve the reader's knowledge of respiratory procedures, equipment, and practice.

The book is arranged according to subject, and each chapter contains multiple-choice questions followed by "explanatory answers" that identify the correct response and give an explanation and reference. A list of references appears at the end of the book, which provides the reader opportunity for further study. This arrangement is easy to follow, and providing answers and references at the end of each chapter is very convenient.

In the Preface, the authors state that the book provides experience with the written testing process and format used by the NBRC. However, the question format is not consistent with NBRC guidelines. Some questions offer three or four options, while others offer five. Several of the multiple-multiple type questions offer "all of the above" or "none of the above" as options, which can be confusing. In some instances, it is not clear whether the authors intended to use a multiple choice or multiple-multiple choice format. For example, Question 577, Page 180, reads

> Pulmonary artery diastolic pressures may be used to estimate pulmonary artery wedge pressures in the absence of
>
> A. mitral stenosis
>
> B. pulmonary emboli
>
> C. tachycardia
>
> D. both a and b
>
> E. all of the above

Numerical ranges often overlap, which makes it difficult to discriminate between options, such as in Question 308, Page 101, which reads

> Running a simple mask at the appropriate liter flow will result in an F_{IO_2} between
>
> A. 0.30 and 0.50
>
> B. 0.40 and 0.60
>
> C. 0.50 and 0.70
>
> D. none of the above

Punctuation is not consistent with NBRC format, and numerical answers are not always listed in ascending order. Trade names for drugs are not always capitalized and generic names are not included (eg, Questions 177 and 178 on Page 60 and Questions 515, 517, and 518 on Page 138). Multiple misspellings exist,—eg, pseudomonas eruginosa (Page 72), heprin (Page 59), and Karposi's sarcoma (Pages 180,193, and 194).

The authors address the majority of the content areas covered on the NBRC entry-level examination; readers are tested on most areas of respiratory care, thus enabling them to identify areas of strength and weakness. Most of the questions, however, are written at the recall level, while 72% of the questions as seen in the NBRC CRTT Study Guide matrix are at the application and analysis level. Also, many of these recall-level questions are trivial and are not written at the same level of difficulty as the current entry-level exam—such as Questions 7, 172, and 367 (Pages 2, 59, and 111, respectively). For example, Question 172 reads

> Which of the following abbreviations means "with?"
>
> A. $\bar{a}$
>
> B. aq
>
> C. $\bar{s}$
>
> D. $\bar{c}$

Very few case study-type questions are included, and, as a result, readers' application, analysis, and decision-making abilities are not adequately assessed.

The list of references is fairly complete though out-dated (containing few recent textbooks or journal articles). For example, the authors use a 1975 edition of Shapiro, Harrison, and Trout's **Clinical Application of Respiratory Care**, a 1973 edition of Egan's **Fundamentals of Respiratory Therapy**, and NFPA pamphlets published in 1968. It is not likely that these texts would be readily available to current students or graduates preparing for their entry-level exams.

This book may be useful as one part of a credential preparation program because answering any number or type of multiple-choice questions can provide good practice. Readers must keep in mind that, although the book may help organize their study program by identifying areas of strength and weakness, the questions are not as difficult as those found on the current entry-level examination. Candidates relying solely on **Respiratory Therapy Examination Review** to prepare themselves for the exam may be at a disadvantage.

Kim L Valeri MSEd RRT
Instructor
Dept of Respiratory Therapy
Medical College of Georgia
Augusta, Georgia

QuitSmart Stop Smoking Kit, by Robert H Shipley PhD. Illustrated soft-cover book (91 pages), 30-minute audiocassette, and Better Quit® Cigarette Substitute. Durham NC: JB Press, 1992. $24.95 plus $3 shipping—QuitSmart, PO Box 4843-RT, Duke Station, Durham NC 27706. (919) 644-0736.

Robert Shipley is Director of the Duke University Medical Center Quit Smoking Clinic and Associate Professor of Psychiatry and Chief of Psychology at Duke University. He has developed smoking cessation programs for several international

corporations, has written three books and numerous articles on the subject, and, perhaps most important, is an ex-smoker. Dr Shipley adequately addresses the problems of physical addiction, habit, and emotional dependence in his book, which contains three phases or chapters: Preparing to Quit, Quitting, and Remaining a Nonsmoker. The author suggests frequent referral to the manual, which is made easy by its pocket size.

The kit contains a manual, **Quit-Smart: Stop Smoking with the Nicotine Skin Patch and Other New Methods,** an audiocassette tape, "QuitSmart Hypnosis," and the Better Quit® Cigarette Substitute. In the first chapter, Preparing to Quit, Dr Shipley invites the user to complete a checklist of reasons for quitting, as well as a 2-item questionnaire to determine whether the individual is physically addicted to nicotine. Dr Jed Rose, inventor of the nicotine skin patch, authors a 9-page section on the patch in question-and-answer format. In this section, the reader is referred to an appendix on nicotine polacrilex gum (Nicorette®). Dr Shipley addresses the issue of switching brands to reduce addiction, listing brands of cigarettes grouped by nicotine content. Users can follow his easy-to-understand guidelines to gradually reduce their nicotine intake until they reach a level where they are ready to actually quit smoking. The chapter concludes with tips on reducing caffeine intake, eliciting support from family and friends, and avoiding smoking temptations. The consumer is asked to sign a Stop Smoking Contract with a quit date.

Chapter 2 begins with congratulations for reaching this point—the interactive and supportive nature of the manual is a strong point. Dr Shipley speaks to reducing stress through relaxation, proper nutrition, and exercise. He introduces the hypnosis tape at this point, and proposes a schedule for its use. The tape is accompanied by instructions and precautions for its use. Side 1 is to be used when quitting, and Side 2 is designed to be used to remain smoke-free. The narrator, Robert Conroy, has a calming voice that I find very relaxing in itself. The Better Quit cigarette substitute, which is included in the kit, is also introduced. This device looks and feels like a real cigarette, and is designed to break the pattern of associating smoking with the nicotine "rush." The filter is adjustable so that the smoker can adjust airflow. The author encourages the consumer to develop a nonsmoking self-image and to keep withdrawal symptoms in perspective. Users are asked to record in the manual the positive changes that they are experiencing after being cigarette-free for several days. Resources such as the National Cancer Information Service and the American Lung Association of Washington DC are provided for the consumer—another "plus."

Chapter 3, "Remaining a Nonsmoker," offers encouragement to the new nonsmoker, from rebuttals to offers of a cigarette to "staying on the wagon." A second contract addresses continuing a smoke-free lifestyle and is followed by tips on weight control and a reiteration of the dangers of smoking and the benefits of not smoking. Lastly, the nonsmoker is asked to list effective coping strategies and the 6-month anniversary of being smoke-free, with a few lines for deciding how to celebrate.

The QuitSmart Stop Smoking Kit is easy to use, interactive, and informative. The manual offers numerous good suggestions to facilitate the process of smoking cessation, with lots of encouragement. The section on the nicotine patch is especially informative and necessary in light of the recent increase in media attention given to the patch. The accompanying audiotape and cigarette substitute are useful supplements to the manual. I enthusiastically recommend the kit to patients who want to stop smoking. Supplemental materials and seminars are available for the health care professional and may be ordered from the address or telephone number listed above.

Crystal L Dunlevy EdD RRT
Assistant Professor of
Respiratory Therapy
The Ohio State University
Columbus, Ohio

Universal Precautions Policies, Procedures, and Resources, edited by Gina Pugliese RN MS CIC, with Contributing Editors Patricia Lynch MBA RN CIC, and Marguerite M Jackson, MS RN CIC. Soft-cover, 400 pages. Chicago: American Hospital Publishing Inc, 1991. Member price $59.95; nonmember price $69.95. Call 800-242-2626 to order.

The editor, Gina Pugliese, is Director of Infection Control and Environmental Safety for the Division of Quality Control Management of the American Hospital Association. Ms Lynch is the Coordinator of the Epidemiology Department at Harborview Medical Center in Seattle, and Ms Jackson is the Director of the Medical Center Epidemiology Unit at the University of California Medical Center in San Diego.

Universal Precautions are federally mandated by the Occupational Safety and Health Administration (OSHA) as a means of reducing the risk of transmission of blood-borne infections in the health care setting. The intent of this manual is to provide health care facilities with a resource to use as they develop or revise policies or procedures involving Universal Precautions. The manual provides the reader with sample policies and procedures from 24 health care facilities and associations, including large teaching facilities,

long-term health care facilities, and small hospitals.

Each facility has provided sample policies and procedures relating to different aspects of a Universal Precautions program, and the editor has produced a well-organized, easy-to-read manual to guide individuals developing policies and procedures for implementing such a program.

Chapter 1 introduces the history of isolation practices and the steps involved in implementing a program.

Chapters 2-7 present various aspects of such a program—Universal Precautions and Isolation Systems, Medical Waste Management, Implementation Plans, Compliance Monitoring, Training and Education, and Employee Health. Each chapter begins with a short introduction and then provides actual samples of policies and procedures from contributing health care facilities.

Chapter 8 introduces OSHA and presents documents from OSHA regarding the management of occupational exposures to blood-borne pathogens. Samples of department-specific tasks and barrier protection are included.

Chapter 9 provides a discussion of various aspects of protective barriers and practices.

Chapter 10 is titled Resources and is exactly that. The chapter contains key reference articles, including the Centers for Disease Control Guidelines on Universal Precautions. Sample posters, pocket cards, and brochures from contributing facilities are included.

Ms Pugliese wanted this manual to reduce the need to "reinvent the wheel" when it came to developing or revising policies and procedures. She has met her goal. This manual is an invaluable resource for individuals responsible for developing or revising a health care facility's Universal Precautions program or any facet of such a program. Department managers may find this manual a good resource if they are responsible for developing department-specific procedures and policies; the sample materials provided may be able to be scaled down to fit the needs of respiratory care departments. However, I do not see this manual as being necessary reading for respiratory care practitioners.

Virginia C DeFilippo MS RRT
Clinical Manager
Department of Respiratory Care
Yale-New Haven Hospital
New Haven, Connecticut

Pulmonary Function Testing: A Practical Approach, by Jack Wanger MBA RCPT RRT. Soft-cover, 227 pages, illustrated. Baltimore: Williams & Wilkins, 1992. $27.00.

According to the Preface, this book "is intended to be both a practical textbook for students and a reference for those who are performing the tests"—cardiopulmonary technologists, respiratory therapists, nurses, and industrial hygienists. In Wanger's opinion, many books now on the market "miss the mark and are intended for the physician or technical director."

As I first opened the book, I had the feeling that the book would be lively. The illustrations are well done and, for the most part, make their point crisply. The print seems larger or, at least, the space between the lines seems greater than in some other texts; thus, it is a book that is easy to read in a train on the way to work. Creative use of font styles and sizes and use of boldface type lend a liveliness to this book when compared to some others in the field that have a gray, somewhat boring appearance.

Five chapters comprise the book: 1. Forced Spirometry, 2. Lung Volumes, 3. Single Breath Carbon Monoxide Diffusing Capacity, 4. Exercise Tests, and 5. Methacholine and Histamine Bronchial Provocation Tests. In keeping with the needs of the target audience, the author has excluded less commonly used tests such as control of breathing and gas distribution. Furthermore, blood gas analysis and sampling are not presented because they are "well covered in other publications."

The book has several attractive aspects. (1) Its appealing simplicity: The student or practitioner who is interested only in the most frequently used diagnostic tests finds them in the first three chapters. (2) Exercise and bronchial provocation are treated in an application-oriented fashion with only the minimal amount of theory included. (Of course, there is a trade-off when one opts for simplicity—the more simplistic, the more application- or nuts-and-bolts-oriented the presentation, the less depth of understanding may be imparted. But, whether today's students are underexposed to theory and are thus undereducated is a philosophical discussion for another time.) (3) Case studies are included at the end of each chapter—no need to hunt for them at the end of the book—and references are cited within the text with a reference list included with each chapter, allowing easy reference tracking.

I opened the book full of anticipation but was met with what I shall call a series of pluses and minuses. For example, the glossary is right up front where it should be, however, no units of measurement accompany the abbreviations. The symbol PB is defined as barometric pressure without an indication of the units used in the book—mm Hg? torr? cm H_2O? atmospheres? The symbol MVV is defined as maximal voluntary ventilation, but what is the standard?—L/min? mL/s? Certainly for some practitioners supplying the units would be specifying the obvious, but for students and other practitioners, this is a necessity. Other examples of this omission are found in some of the equations in Appendix I and elsewhere—a small

point perhaps, but important to the would-be user.

Some important information is omitted—perhaps in the interest of simplicity. In Chapter 1, quality control procedures of spirometry are suggested, but the details (ie, checking timing, assuring reproducibility, and calculating means, standard deviations, and coefficients of variation) are not supplied to allow the uninitiated reader to acquire and use the data.

On Page 24, the author presents an excellent section on patient preparation prior to spirometry, but fails to explain the importance of actually measuring the patient's height and weight rather than relying on self-reports.

Photographs of equipment are frequently paired with line drawings—sometimes very effectively (as in Figure 1.6 on Page 8, a photograph of a Stead-Wells spirometer paired with an elucidating line drawing) but sometimes not, as that of the diaphragm-type spirometer (Figure 1.13 on Page 13), which does not help me understand how that type of spirometer operates.

Some of the explanations are simplistic but helpful. In Chapter 5, a simplified overview of the physiology of airway responsiveness will likely reactivate memories of terms such as autonomic and sympathetic and thus help one to understand the mode of action of methacholine and histamine in bronchial provocation tests. However, if these are new concepts to the reader, the explanation of the nervous system may convey little, if any, understanding. Another example is found on Pages 5 and 6, where equal-pressure-point theory is explained. If one is already familiar with this theory, then the explanation makes sense; otherwise, it may be of little value.

One particularly disappointing feature of the book is the glut of typographical errors and imprecise wording—for which the editor would seem as responsible as the author. An errata sheet has been prepared to accompany the book.

One final point—although it may be apppropriate for the author to offer suggestions and advice from his own experience, I believe that his citation of unpublished work related to the effect of bronchodilator on diffusion capacity and the minimal resting HbO_2% for an exercise test puts him on shaky ground.

In summary, I read this book with the intent of listening closely to what the author had to say. I read it with the intent of learning, but, to my disappointment, the errors caught my attention and got in the way of my learning. The author undertook a difficult task—trying to make a complex topic less complex—for which he should be applauded. However, a textbook such as this is likely to be widely read by persons who have not yet acquired the knowledge necessary to "sort the wheat from the chaff." Such a text should be more carefully reviewed for errors than this one apparently was. I hope that another edition will follow. I am certain that we will see major improvements.

Louis F Metzger RPFT
Center for Sleep &
Respiratory Neurobiology
University of Pennsylvania
Medical Center
Philadelphia, Pennsylvania

Atlas of Radiology of the Chest, edited by Dame Margaret Turner-Warwick DBE DM PhD, Margaret E Hodson MD MSc, Bryan Corrin MD, and Ian H Kerr MA MB BCh. Hardcover, 155 pages, illustrated. London: Gower Medical Publishing, 1991. $70.00. Distributed in the USA and Canada by JB Lippincott Co of Philadelphia, Pennsylvania.

As the editors note in the Preface, this work is not a complete textbook of chest radiology but rather an overview of a wide range of chest conditions for use by physicians and allied health professionals. It is, by their admission, inevitably incomplete and they humbly apologize for any omissions.

The 9 chapters, beginning with a 22-page review of normal chest radiography cover every type of chest radiographic procedure except MRI. The first chapter is a thoughtful way to begin the book for the reader who is beginning his/her exposure to chest radiology and for those of us who need a refresher. In subsequent chapters, diagnosis of a mass, tumors, diseases of the airways, infection and allergic disorders, vascular diseases, occupational lung diseases, diseases of the mediastinum, and chest injuries are covered. Despite its incompleteness, the 9 chapters cover numerous common and uncommon chest diseases and diagnostic radiography procedures. Accompanying each reproduction is a short paragraph describing it and the pathology. In some cases a hand-drawn diagram is presented to better illustrate the actual picture. Most of the reproductions are quite clear, although a few are of poor quality.

This volume is a quick look at many respiratory diseases, and is a reasonable chest roentgenogram reference for the student and practitioner (see recommendations). Although the editors have attempted to provide an affordable resource, **Atlas of Radiology of the Chest** is not, however, a challenge to its bigger brothers, which serve as in-depth, thorough references to chest radiology and cardiopulmonary disorders.

I thought a lot about who this book might best suit, and decided to recommend it to the chest text collector, radiologist, and local medical library. However, the book does not go far enough or deep enough for me

to recommend it to the student for personal use or to the average practitioner as an adequate reference. Certainly better, more effective, though possibly more expensive, books are available; I direct the reader to the following books as an alternative: **Handbook in Radiology: Chest Radiology**, by Mosby Year Book, fits in the pocket and, although it contains no figures, is surprisingly complete at $24.95 and/or **Roentgenology of the Chest**, edited by Felson, as the high dollar, voluminous "gold standard" of chest radiology. In a market flooded with resources, I do not feel **Atlas of Radiology of the Chest** brings anything new, unique, or different to bear.

Douglas B Eden BS RRT
Respiratory Care Practitoner
Fairfax Hospital
Fairfax, Virginia

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Letters

ACLS Skills for the Respiratory Therapist

In response to the editorial by our colleagues, Drs Barnes and Durbin, in the June issue of Respiratory Care,[1] I would like to state that in my opinion their recommendation should be acted upon immediately by the profession. I have been wondering for years why Advanced Cardiac Life Support (ACLS) and/or its pediatric and neonatal variants are not mandated for respiratory care practitioners (RCPs) by the respiratory care educational establishment. It is obvious to me that an RCP who has a primary role in resuscitation is as likely a candidate for this advanced training as are the nurses in the operating suites my wife runs in her job as surgical coordinator for an outpatient surgery center. I think we have lost some professional credibility as a group by not wholeheartedly having RCPs (at least those at the RRT level) certified en masse.

Any nurse can take these courses, and the nursing profession has jumped at the chance to upgrade their skills (sometimes resulting in an increase in pay). Although the primary reason should not be to make more money, I am not opposed to opportunities for enhancing my knowledge and skills that also increase my earning power. The self-esteem to be gained by achieving ACLS certification deserves mention also. Yes it's hard, but so what? The Registry exam I took in '86 was harder, at least to me. ACLS skills should be learned in RCP programs, not in postgraduate courses. The new RCP should come to the job already certified. The fact that this has not been done seems to be a serious mistake in the planning and vision for my chosen profession. We are the people who drop what we're doing to respond to a Code. We are the people who attend every Code. As members of the profession we should be familiar with everything that can be medically done in a Code. Our profession should be the one that teaches ACLS to other providers.

Christopher Horn RRT
President
Advanced Home Respiratory Care
Orange Park, Florida

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ACLS—One Department's Experience

I read with interest the ACLS mandate editorial.[1] Since January 1990, the Respiratory Care Department of St Luke's Episcopal Hospital has been responsible for coordinating ACLS classes within our institution. These courses are a collaborative effort with our medical staff, cardiology fellowship program, nursing, respiratory care, and other ancillary care professionals. This came about as a result of a need for in-house training at a time when our department had the resources to accommodate the need—we have several critical care therapists who are ACLS Providers and Instructors.

Because our department is responsible for the ACLS classes, the classes are indeed accessible to our own therapists as well as to therapists from around the city. Although the greatest number of students continue to be physicians and nurses, I have seen an increase in the number of RCPs who are registering for the classes—and passing.

Our therapists are reimbursed for the course if the pass. If they do not pass, they are allowed to take the next course free of charge. This is a great incentive to take and pass the class, and successful completion is also reflected on their yearly Performance Evaluations.

The respect our department has achieved from providing an important training class to therapists, nurses, and physicians is immeasurable. Thank you Dr Barnes and Dr Durbin for addressing this issue!

Elizabeth F Bearden RRT RCP
Coordinator
ACLS Program
St Luke's Episcopal Hospital
Houston, Texas

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More on ACLS Training

I have a great appreciation for the comments published by Thomas Barnes and Charles Durbin entitled "ACLS Skills for the Respiratory Therapist: Time for a Mandate."[1] I agree wholeheartedly that respiratory care practitioners (RCPs) who are members of resuscitation teams should obtain Advanced Cardiac Life Support (ACLS) credentials for the reasons they stated.

One of the goals of our respiratory care department is to have all RCPs who perform endotracheal intubation to attain ACLS certification. There is a method for accomplishing this that is more cost-effective than paying fees for ACLS certification and recertification courses. Several of our supervisors have completed ACLS instructor training and are able to conduct the courses under the direction of our medical director, who is a Physician Instructor. We have purchased some of the necessary equipment and bor-

row the remainder. ACLS certification and recertification courses are offered at least semiannually for RCPs in our department. Certification by this method results in substantial savings, with the added benefit of an increase in job satisfaction for the ACLS Instructors. The ability to complete the training on familiar turf also helps to eliminate anxiety for participating RCPs.

This method may not be practical for every respiratory care department but it has worked well for us, and I recommend it for other large departments.

Susan Rinaldo Gallo MEd RRT
Clinical Coordinator
Respiratory Care Services
Duke University Medical Center
Durham, North Carolina

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Dinosaurs, RCPs, and ACLS

The June issue of RESPIRATORY CARE brought quite a surprise to me—with no fewer than four references to the need for RCPs to achieve ACLS completion and quite a bit of discussion about the difficulties in getting into an ACLS course and, once accepted, of passing the course.[1]

Our Respiratory Care Department has been building toward total ACLS completion for the last 7 years, and our goal was achieved more than a year ago. My surprise relates to why most RC Departments have not done the same. I am not saying that ACLS is not difficult; it is. But, it is certainly a 'doable' project if you plan properly, are totally committed, and take proper steps to ensure that your staff is as committed as you are.

Maybe the best way to explain why I think total ACLS completion is so obtainable is just to tell you how we managed to make it happen in Lufkin, Texas.

It all began back in 1982 when there were still a few dinosaurs roaming the earth. My department was having problems keeping our BLS cards current. The reason? Only one person taught BLS for the entire hospital, and it was impossible for this individual to satisfy the needs of the whole hospital.

I was sitting and wondering who I could get to teach BLS to our staff, when one of those dinosaurs began playing hopscotch outside my window. All that ground shaking jarred my brain out of its rut, and I had a revelation. Some of the RCPs could become instructors and teach BLS to the other RCPs—anytime we wanted.

Well, we did just that. Shortly thereafter some other hospital workers saw our classes going on and asked "Can we play too?"

Being the social 'bugs' that RCPs are known to be, we said sure. Soon the few non-RCPs we let into our classes grew to a flood. There were more employees wanting BLS classes than we had instructors to do the classes. After sitting through a few more hopscotch games, another light dawned—Why not make all RCPs BLS Instructors and make a few RCPs BLS Instructor-Trainers? We would then be self-sufficient. However, this move caused a few problems. Teaching a BLS class required confronting the most dreaded of all monsters, Public Speaking. It also meant that RCPs would have to correct registered nurses and doctors in BLS classes if they performed CPR incorrectly. In the land of the RC Department, there went up a great wailing and gnashing of teeth. RCPs turned green, tossed their cookies, and fainted dead away—all in an effort to avoid the above monsters. But we put our heads down and went on. Every RCP was loaded into the breech, the hammer cocked, and the trigger pulled. And, as in all RCP legends, they all rose to the occasion. Not one RCP died, like they thought they would, and after they had taught a few courses, it did not seem so bad. Many began to wonder what they had been scared of. It was like finding that the boogeyman who lives in your closet or under your bed is not so tough after all. There was another benefit also (although sometimes I wonder if it always is a benefit) the techs began to mutate—because they had to speak in CPR class they soon decided to began speaking elsewhere. They started telling doctors about the patients they were caring for and making suggestions to improve their care; they talked to other health care professionals on an equal footing instead of 'up to.' Basically, the RCPs were thinking of themselves as professionals and demanding that they be treated that way.

The RC Department did so well teaching CPR to our employees that we were asked to teach CPR for the entire hospital. Being the voracious little workers that we are, we accepted.

Well, one thing led to another. If we could do so well with BLS, why couldn't we do the same with ACLS? Again in the land of the RC Department, there went up a great wailing and great gnashing of teeth. This was 1984 and only doctors and RNs took ACLS because they had to read EKGs, give drugs, and shock people. Surely we could not expect RCPs to do this. But, then, after the dust had settled, RCPs began to pass ACLS—most the first time attempted. And the RCPs who had to take ACLS first made sure that the other RCPs had to attend too—only because they cared so much about their fellow workers' educational status, of course.

Now, 6 years later, all RCPs are expected to take ACLS, and 100% of our full-time staff have passed ACLS. Somewhere along the way the RC Department picked up the responsibility for organizing and teaching ACLS countywide. Thankfully, some other great professionals wanted to help: RCPs are actually not the largest group of ACLS instructors in Lufkin: we have many excellent nurses, paramedics, and doctors who help provide ACLS for our entire county.

And wouldn't you know it, passing ACLS changed the RCPs too. On Codes, they speak up when something is wrong because they know what is correct and proper. When they look into the mirror, they see a professional, not just an employee—and know that professional was created by their own ability.

This scenario might sound like a cake walk or that you can do it alone. Neither are true. The only way it happened here was through the efforts of a superb Assistant Director, an excellent physician who helped push it, and an Administration that believed education was important and backed us financially. Also, we had one of the best Nursing Departments around—that worked as hard as we did to see ACLS start and grow. And, finally, we had a life-saving secretary who (using her own kind of ACLS) miraculously managed to keep the tons of paperwork flowing in the right direction.

In conclusion—as great as ACLS is for patients, its implementation is not for the faint at heart. But, then, no one ever said RCPs were faint at heart.

I will close now, because I think I hear a dinosaur playing hopscotch outside.

David Henson RRT
Director, Respiratory Care
Memorial Medical Center of
East Texas
Lufkin, Texas

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Emergency Care and ACLS

The June and July issues of RESPIRATORY CARE[1,2] on emergency respiratory care should be required reading for every person who accepts a salary for working in respiratory therapy, including medical directors and educators.

Drs Barnes and Durbin's editorial calling for ACLS certification for respiratory therapists[3] comes at a strange time in our professional history, as do Dr Kacmarek's comments regarding endotracheal intubation.[4] That is, after so many years of being expected to be silent, obedient "beggars" and "go-fers" at resuscitations, many therapists have become ACLS certified and also have been intubating for some time now. Yet in recent years a backlash has evolved in some hospitals. Therapists who had been safely and successfully intubating are now being told that they can no longer do so, and other institutions will not even consider letting therapists intubate. Have we been going backwards professionally?

This is not merely regional observation. I have worked in respiratory therapy in four states, and I have been a department manager in various-sized institutions, ranging from an 80-bed rural hospital isolated in the Mojave to one of the world's largest, most prestigious medical centers in Baltimore. Quite frankly, except for the research opportunities available at the larger place, I have had a lot more fun at the smaller hospitals. We routinely intubated, and we ran our own ventilators within broadly defined physician orders that showed respect for our intelligence and professional knowledge. The question asked by the medical staff at such hospitals is not, "Would you like permission to intubate?" but rather "How well do you intubate? Because you're going to have to do it here, and do it a lot." Also, a review of a respiratory therapy publication that features national help-wanted ads[5] shows that in many of those advertisements a recruiting point is made of the fact that "we intubate here."

So therapists are intubating and have been for some time ... in some hospitals. What has happened in the other institutions? In hospitals affiliated with medical schools the answer is usually that a major *raison d'être* is the training of doctors; so, interns and residents do a lot of the procedures that in nonteaching hospitals are done by respiratory therapists and critical care nurses. This is one area where the 'grunts'—the skilled, expert therapists who actually do the procedures at nonteaching community hospitals—should be invited to participate in seminar discussions about what RCPs should or should not be doing. I have found this to be a disappointingly rare occurrence at state and national association meetings. Comments from the majority who make up the real world of respiratory care would be a refreshing change.

Okay, so what about those hospitals where therapists used to intubate and are now no longer allowed to do so? It has been my experience that less-than-sincere reasons are usually given for such prohibitions, reasons that include alleged concerns about liability, skill, training, education, etc. Sorry, but such verbiage displays a rather insulting, parochial ignorance of who and what we are, and it also completely disregards what I've mentioned above: We already are intubating at many hospitals, and we do it well. This would seem to ac-

centuate a rather obvious point: Those same concerns about liability, training, etc have already been addressed by progressive, caring medical directors and hospital administrators at places where therapists have been intubating for years. So the question isn't "Should our medical directors support our efforts to intubate?" but rather "Why have some medical directors (and administrators) been offering such support all along, and what possible real objections can be offered by other physicians for opposing us on this issue?" The too-often unmentioned financial reasons for prohibiting therapists from intubating should ethically not be part of this decision, and it would be interesting to see that subject discussed in an unbiased manner.

Those who advocate "only doctors should intubate" are also ignoring the irony that many of those endotracheal tubes that we ultimately attach to our ventilators were inserted by paramedics. Surely then, well-educated, well-trained critical care respiratory therapists, airway experts—Well aren't we? Isn't that what we claim?—should be doing as much. As pointed out by Kacmarek, our paramedic brethren, often working under terrible conditions out in the field, have a greater than 90% success rate in getting that tube into the trachea.[4] I can think of no more potent argument for countering objections to therapists intubating.

Tom Burns RRT
Instructor in Respiratory Care
GateWay Community College
Phoenix, Arizona

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Nothing New but Still Useful: F_{DO_2} Estimation

Thank you for the letter from Dr Atlas[1] on calculating F_{DO_2} for mixtures of air and oxygen.

I first used these formulas in 1972 when I started my respiratory care practitioner career at Massachusetts General Hospital. At the time, the Emerson Postoperative Ventilator came with tables and literature for estimating the titration necessary to achieve specific oxygen concentrations. I reworked the formulas in a manner identical to that described in Dr Atlas's letter to solve for oxygen flow and have used these successfully for many years. The formulas have since appeared in McPherson's third edition,[2] and presumably in the earlier editions as well.

We currently use these formulas most commonly to set air and oxygen flows for a particular F_{IO_2} and total flow when using the Bio-Med MVP 10 Infant Transport Ventilator.

Charles Kettell RRT
Neonatal Pediatric Supervisor
Division of Pulmonary Medicine
Maine Medical Center
Portland, Maine

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BiPAP in the Acute Care Setting

A device (BiPAP, Respironics Inc, Monroeville PA) designed for nocturnal ventilatory assistance in the home has become increasingly popular in the acute care setting.[1-4] The device consists of a nasal CPAP blower coupled with a solenoid system that provides timed cyclic delivery of positive airway pressure at two different levels.[5] The device has been popularly referred to almost as though it were a unique mode of ventilation. However, one can easily classify this device using Chatburn's method of classification.[6] The machine can be classified as patient- or time-triggered, pressure-limited, and time-cycled. Newer models allow patient triggering. Thus, as you can see, BiPAP is really not a new mode of ventilation.

A number of studies have shown the benefits and efficacy of nasal volume ventilation for patients with chronic respiratory insufficiency,[7-16] and BiPAP may be an effective and reasonable alternative to such volume-cycled devices in the home. The advantages of the BiPAP device are said to be that it is small, portable, and lightweight, relatively inexpensive to purchase or lease, quiet and easy to maintain, easy to apply and remove; patients seem to tolerate it well; and it is a noninvasive method of augmenting a patient's spontaneous ventilation. In a small study by Strumpf et al,[5] the BiPAP device was shown to be as effective as nasal volume ventilation in the patients studied—patients suffering from postpoliomyelitis syndrome, muscular dystrophy, osteogenesis imperfecta and kyphoscoliosis, and chronic bronchitis—who had all previously been on volume ventilators or negative pressure ventilators nocturnally.

However, I have observed, firsthand and from anecdotal reports, what I call a disconcerting shift in the way BiPAP is being used in the acute setting—as a crisis intervention tool. Patients on the verge of intubation or patients recently extubated whose respiratory status is vola-

tile have been placed on BiPAP to stave off intubation.

The units have their disadvantages. The BiPAP unit is a pressure-limited device, and therefore suffers the shortcomings of all pressure generators. The volumes delivered by the BiPAP device vary with changes in lung compliance and airways resistance. The device has also reportedly been less likely to control hypoventilation if pressures exceeding 25 cm H_2O are required.[1] Although newer models of the BiPAP device come equipped with backup rates in case of bradypnea, and "hospital"-equipped machines provide volume and pressure alarms, it is important to realize that these backup rates and pressures may not be adequate to ventilate the patient. Strumpf et al[5] have recommended that BiPAP not be used "in patients who are ventilator dependent, or who have very limited ventilatory reserve," and the manufacturer states on Page 1 of the clinical manual that the BiPAP machine is meant to augment a patient's spontaneous ventilation and is not a life support ventilator.[17] In the recent literature, there have been a few cases of what the investigators report as successful avoidance of intubation by BiPAP. The sample sizes have been small, 1 to 39 patients, and most reports are in abstract form at this time.[1-4] It certainly makes sense that patients in acute or impending respiratory failure would benefit from the application of positive airway pressure to augment their ventilation. We would seek to decrease their work of breathing, normalize their P_{aCO_2}, and improve their pH. That is why we intubate them. However, I have seen BiPAP used much like IPPB was used in the past—an hour on BiPAP, an hour off. If the blood-gas results are poor, the patient is returned to the BiPAP. Does this help the patient? Or, does it prolong the time from when patients should have been intubated to when they are intubated?

The nature of the BiPAP device does require some degree of patient cooperation and tolerance. I have seen patients on the verge of intubation during a BiPAP trial who were not able to trigger the machine into inspiration in a consistent way, probably resulting in an increase in work of breathing. It is my belief that if a patient needs his ventilation augmented acutely, he should be intubated, fully understanding the risks and complications of endotracheal intubation. I believe that the risks associated with insufficient ventilation and oxygenation may be greater than those associated with intubation. I question whether a patient is better off remaining extubated for a period of time, struggling with nasal mask ventilation and perhaps with an unresponsive ventilator, instead of being intubated. Is it better to be extubated and be on BiPAP for 6 days, on the verge of reintubation? Or is it better to remain intubated for, say, 3 more days, and be well enough to breathe on your own? We certainly don't want to wean from 10 breaths to 6, to 4, to 15 of IPAP, to 10 of IPAP, and on and on. As was also stated earlier, the machine has a backup mode, but just how effective is it in providing adequate ventilation for the patient? I think the answer to this question is patient specific, and I would be leery of depending on it in all cases. The patients we are talking about are close enough to being intubated or reintubated that they may easily have acute changes in compliance or resistance that render the machine's assistance inadequate. These patients may also reach the point at which they can no longer protect their airways and are at an increased risk of gastric insufflation, vomiting, and aspiration—progressing even to the point of respiratory arrest. It may be argued that there are enough monitors to prevent a patient from progressing to a point at which he suffers serious harm. I contend however, that we are obliged to adequately ventilate and oxygenate patients before any physiologic sequelae occur. I cannot stress enough, as does the manufacturer, that BiPAP is intended to augment a patient's spontaneous respiration—not to be a life support ventilator.

It is my belief that BiPAP should be used only in a select group of patients until there is more literature supporting its use in the acute setting—patients with skeletal or muscular defects who benefit from nocturnal ventilatory assistance, patients with severe COPD who benefit from long-term nocturnal ventilatory assistance, patients who cannot tolerate conventional CPAP therapy who may benefit from a BiPAP device (anecdotal evidence). At this point in time, our goal with the use of BiPAP should be to improve the patient's daytime function either by alleviating nocturnal hypoventilation or resting the ventilatory muscles at night.[5]

The BiPAP units certainly have their place in the home care setting. Whether it is appropriate for them to become more common in the acute setting needs to be determined by more extensive study. While carefully structured clinical trials with meticulous data collection are underway, it is our obligation to our patients to recognize the limitations of noninvasive ventilation and to use the devices and adjuncts as they were intended.

William P McGarry III BS CPFT RRT
Respiratory Care Supervisor
Department of Respiratory and Critical Care
Beth Israel Hospital
Boston, Massachusetts

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Could You Be The Reason for a "Spider in the Airway?"

We report this phenomenon because to the best of our knowledge it has not been reported before. This spider (or other insect), which was dead and organized in a web (Fig. 1), was discovered by one of our anesthesiologists during routine preparation of a breathing circuit (from a pediatric circle system, Fig. 2) prior to starting a case. The manufacturer was notified of the incident.

Fig. 1. Dead spider organized in web within breathing circuit.

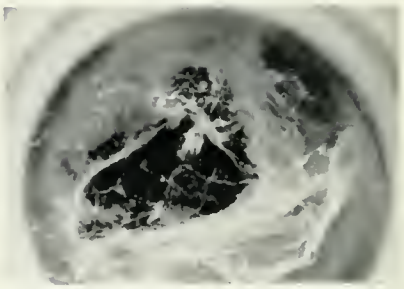

Fig. 2. Dead spider organized in web as seen from exterior of breathing circuit.

We report this finding to caution all practicing anesthesiologists, anesthetists, and our respiratory care colleagues who handle prepackaged durable items.

Mary C Theroux MD
Michael Zagnoev MD
Carmen Ponce CRNA
Alfred I duPont Institute
Wilmington, Delaware

Some History Related to the Sigh

The use of sighs or deep breaths is a much older story than may have been suggested by the authors of Kittredge's Corner in the May issue of the Journal.[1]

The use of sighs to counteract atelectasis in polio patients ventilated in iron lungs was discussed by Dr MB Visscher at a National Infantile Paralysis Foundation Round Table on "The Physiology of Respiration and Respirators with Particular Reference to Poliomyelitis"[2] held in Minneapolis, Minnesota, in October 1947.

At a later National Foundation program on Respiratory Problems in Poliomyelitis, held in Ann Arbor, Michigan, in March 1952, Mr John H Emerson[3] described the deep breath attachment for tank ventilators

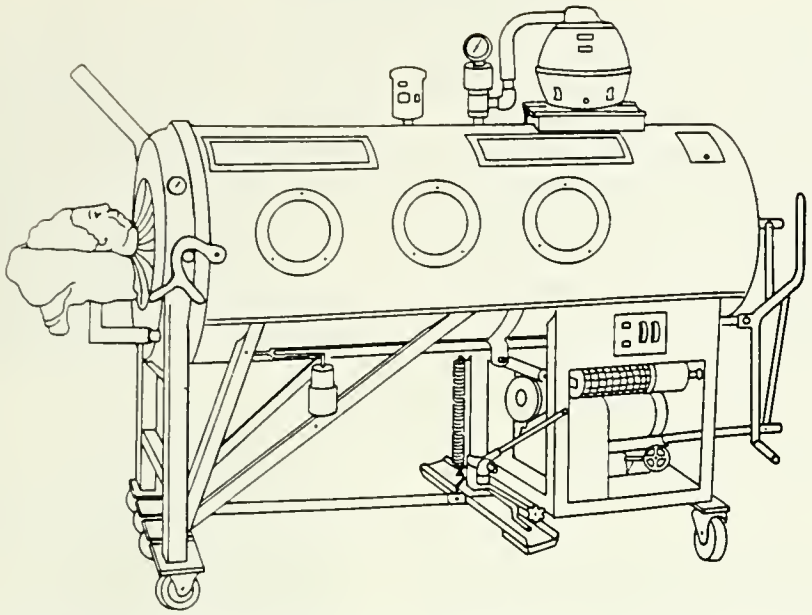

Fig. 1. The The 1952 model Emerson Tank Ventilator fitted with the deep breath attachment—the round device on top of the ventilator.

he had put into production acting upon Dr Visscher's suggestion (Fig. 1). He complained that they had been little used so far. The illustration is reproduced from a photograph in the 1952 Emerson catalogue. It was not until 11 years later when Bendixen and colleagues spread knowledge of Mead and Collier's work in anesthesia and respiratory circles that the use of deep breaths became well known.

It was a similar story concerning the introduction of PEEP. In 1955, Jack Frumin and Arnold St Lee designed and manufactured their Auto-Anestheton.[4] This was a CO_2 servo-controlled ventilator that varied the patient's tidal volume as necessary to maintain the preset end-tidal CO_2. Though they could easily maintain a steady arterial CO_2 level, reliable oxygenation in spite of adequate inspired oxygen concentration could not be reliably achieved until 7 cm H_2O of end-expiratory pressure had been added.[5] Their CO_2 servo-controlled ventilator design did not influence ventilator designs and they did not coin a term like PEEP to describe their method. Thus, positive end-expiratory pressure did not become well known until it was rediscovered by McIntyre and colleagues in 1969.[6]

Not only is it necessary to have a good idea, but one must also convince one's contemporaries.

Leslie Rendell-Baker MD
Professor of Anesthesiology
Loma Linda University
School of Medicine
Loma Linda, California

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Branson and Campbell reply:

We appreciate Dr Rendell-Baker's comments on the origin of the sigh breath. Using the information he provided, we attempted to obtain the citations detailing the work of Visscher and Emerson. Despite our rather complete medical library, we were disappointed to find that the publications are unavailable. This appears to be a case of "You had to be there!" We beg Dr Rendell-Baker's indulgence on our failure to mention those.

We agree with Dr Rendell-Baker's statement concerning convincing one's contemporaries. It appears even more difficult to convince ones contemporaries to abandon a good idea that has failed to prove its usefulness. We believe that the time has come to call for the removal of

the sigh-breath option from ventilators—and to allow the sigh to die.

Richard D Branson RRT
Robert S Campbell RRT
Department of Surgery
University of Cincinnati
Medical Center
Cincinnati, Ohio

'Bye Sigh

I received my anxiously awaited May issue of RESPIRATORY CARE on the 11th of the month. The successful launching of Kittredge's Corner[1] (with the slight drawback of the missing table[2]) afforded me a brief moment of pride in accomplishment.

But, that's not what this letter is about. Two days after the arrival of the Journal, I received the familiar blue and white envelope, the trademark of RESPIRATORY CARE, with the Little River, California, return address. I opened the envelope with some anticipation and was not disappointed. In quintessential (Boy, did I want to spell that with a K) Kittredge form was the following ditty, I now reproduce for the enjoyment of others, with Phil's permission.

If a kiss is just a kiss,
And a sigh is just a sigh,
Is it time to kiss,
The sigh Goodbye?

The art of communication through the written word, they say, is lost on this video generation. Yet, I still delight in the creative and clever coupling of words by those blessed with the talent for writing.

As usual, Phil has left me thinking, "Boy, I wish I'd written that." Thanks, Phil. Keep up the good work!

Richard D Branson RRT
Instructor
Division of Trauma & Critical Care
University of Cincinnati
Medical Center
Cincinnati, Ohio

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Notices

PUBLICATION OF INTEREST

Centers for Disease Control. Prevention and Control of Influenza: Recommendations of the Immunization Practices Advisory Committee (ACIP). MMWR 1992;41(RR-9):1-17. [Some excerpts are provided below.]

**

Persons who are clinically or subclinically infected and who attend or live with high-risk persons can transmit influenza virus to them. Some high-risk persons (eg, the elderly, transplant recipients, or persons with AIDS) can have low antibody responses to influenza vaccine. Efforts to protect these high-risk persons against influenza may be improved by reducing the chances of exposure to influenza from their care providers. Therefore, the following groups should be vaccinated:

1. physicians, nurses, and other personnel in both hospital and outpatient-care settings who have contact with high-risk persons among all age groups, including infants.
2. employees of nursing homes and chronic-care facilities who have contact with patients or residents.
3. providers of home care to high-risk persons (eg, visiting nurses, volunteer workers).
4. household members (including children) of high-risk persons.

**

Inactivated influenza vaccine should not be administered to persons known to have anaphylactic hypersensitivity to eggs or to other components of the influenza vaccine without first consulting a physician (see Side Effects and Adverse Reactions).

**

Beginning each September, when vaccine for the upcoming influenza season becomes available, high-risk persons who are seen by health-care providers for routine care or as a result of hospitalization should be offered influenza vaccine. Opportunities to vaccinate persons at high risk for complications of influenza should not be missed.

**

The optimal time for organized vaccination compaigns for high-risk persons usually is the period between mid-October and mid-November.

**

Copies can be purchased from Superintendent of Documents, U.S. Government Printing Office, Washington DC 20402-9325 or via telephone (202) 783-3238.

THE NATIONAL BOARD FOR RESPIRATORY CARE—1992 Examination and Fee Schedule

RRT Examination

EXAMINATION DATE:	DECEMBER 5, 1992
Applications Accepted Beginning:	June 1, 1992
Application Deadline:	August 1, 1992

RPFT Examination

EXAMINATION DATE:	DECEMBER 5, 1992
Applications Accepted Beginning:	July 1, 1992
Application Deadline:	September 1, 1992

Fee Schedule

Entry Level CRTT—new applicant:	$ 90.00
Entry Level CRTT—reapplicant:	$ 60.00
RRT Written and Clinical Simulation—new applicant:	$190.00
Written Registry Only—new applicant:	$ 90.00
Written Registry Only—reapplicant:	$ 60.00
Clinical Simulation Only new and reapplicant	$100.00
Entry Level CPFT new applicant:	$100.00
Entry Level CPFT—reapplicant:	$ 80.00
Advanced RPFT—new applicant:	$150.00
Advanced RPFT—reapplicant:	$130.00

	Active	Inactive
CRTT Recredentialing:	$25.00	$ 60.00
RRT Recredentialing:		
Written Registry Examination	$25.00	$ 60.00
Clinical Simulation Examination	$65.00	$100.00
CPFT Recredentialing:	$25.00	$ 80.00
RPFT Recredentialing:	$25.00	$130.00
P/P SPEC:	$25.00	$130.00
Membership Renewal:		
CRTT/RRT/CPFT/RPFT		$ 12.00

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Calendar of Events

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of the month two months preceding the month you wish the ad to run. Submit copy and insertion orders to: Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane • Dallas TX 75229-4593.

AARC & AFFILIATES

August 26-28 in Little Rock, Arkansas. The ASRC holds its 21st Annual State Meeting and Educational Seminar, "Catch the Arkansas Spirit," at the Excelsior Hotel and Convention Center. Sessions on trauma and heart/lung transplants are featured. Social events include a riverboat cruise and barbecue. Contact Jim Bradford (501) 686-6285.

August 27 in Sacramento, California. The CSRC, Chapter 11, presents its Annual Seminar, "Tools of the Trade," in conjunction with the California State Fair. The seminar, admission to the Fair, dinner at the Fair's VIP tent, and California wine tasting are included in the admission price. Contact Marie Kearney (916) 441-7626 or Mark Goldstein (916) 933-3469.

August 27-28 in Jacksonville, Florida. Chapter 5 of the FSRC presents its 10th Annual 1992 River City Symposium. Topics include ethical, legal, and technical aspects pertinent to current practice of respiratory care. Contact Scott Watterson, Department of Respiratory Care, PO Box 2000, Orange Park FL 32067-9984. (904) 276-8630.

September 17-18 in Tucson, Arizona. The AzSRC hosts its Annual Meeting at the Tucson National Resort, Golf Club, and Spa. Contact Becky Shocklee, 3341 N 31st St #108, Phoenix AZ 85016.

September 17-18 in Bethel, Maine. The Annual Fall Seminar, presented by the MSRC, is held at the Bethel Inn & Country Club. Come get a taste of New England and its beauty in the Fall. Enjoy activities such as an 18-hole championship golf course, tennis, canoeing, swimming, an outdoor barbecue, and much more! Outstanding speakers, and vendors present the latest in available technologies. Contact Edward Amend, 2810 Isthmus Rd, Rumford ME 04276-9724. (207) 364-4581, ext 362.

September 23 AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents "Aerosol Administration," one in a series of live satellite videoconferences titled "Professor's Rounds in Respiratory Care." Featured presenters are David J Pierson MD and Dean Hess MEd RRT. Site registration for entire staff is $245 for AARC members. Call (214) 830-0061.

September 24-25 in Indianapolis, Indiana. The ISRC presents its Fall Conference at the Marriott Inn. The awards ceremony and Sputum Bowl are held on Thursday. Contact Jan Doherty (800) 327-3152 or (219) 761-3607.

September 29-30 in Honolulu, Hawaii. The HSRC holds its 19th Annual Respiratory Care Conference at the Hilton Hawaiian Village Hotel. Contact Helen M Ono RRT, 1717 Palolo Ave, Honolulu HI 96816. (808) 547-9532.

September 30-October 2 in Traverse City, Michigan. The MSRC presents its Annual Fall Conference at the Shanty Creek/Schuss Mountain Resort. The general session focuses on asthma management, critical care, ventilation and waveform interpretation, nutrition, and risk management. Also, the Cardiopulmonary Diagnostics Membership Section presents pulmonary function and sleep disorder testing. Social events include a wine-and-cheese reception, golf outing, cookout, and dance. Come join us for another successful conference, and enjoy the beautiful colors of Fall in Michigan. Contact Beth Hill RRT, Bay Medical Center, Respiratory Care Dept, 1900 Columbus Ave, Bay City MI 48708. (517) 894-3166.

October 2-4 in Key West, Florida. Chapter 1 of the FSRC and Sunset Seminars present the 6th Annual Southernmost Sunset Seminar at the Holiday Inn Beachside. Registration includes a Saturday evening conference aboard the Atlantic X dinner/casino cruise ship. Contact Dave Robbins, Coral Gables Hospital, 3100 Douglas Rd, Coral Gables FL 33134. (305) 441-6819.

October 14 in Auckland, New Zealand. The NZSRC presents its Annual Scientific Meeting at the Aotea Centre. The meeting precedes the Australian-New Zealand Intensive Care Society Conference. Contact Graeme A'Court, PO Box 10148, Balmoral, Auckland, New Zealand. (643) 640640.

October 16 in Long Island, New York. The Southeastern Chapter of the NYSSRC presents its 24th Annual Symposium, "Focus on Technology," at the Marriott Hotel in Uniondale. Speakers include Michael McPeck, Nathan Seriff, Joseph Lore, Steven Marzo, Margaret McGovern, and AARC President Bob Demers, who present "Clinical Hazards Associated with Aerosols." Contact Ken Nugent RRT (516) 444-8511.

October 28-29 in Sturbridge, Massachusetts. The MSRC presents its 15th Annual Meeting and Exhibition at the Sturbridge Host Hotel and Conference Center. Topics include adult and neonatal ventilator management, the RCP's role in smoking cessation, and interpretation of ventilator graphics. Events include Sputum Bowl, golf tournament, and awards banquet. Contact Bill McGarry CPFT RRT, Respiratory Care, Beth Israel Hospital, 330 Brookline Ave, Boston MA 02215. (617) 284-0782.

October 30 in Bear Mountain, New York. The Hudson Valley Chapter of the NYSSRC presents its Annual Educational Seminar, "Challenging Issues in the Respiratory Care Environment," at the Bear Mountain Inn. Topics include patient assessment, infection control, and risk management. Contact Larry Lyman (914) 358-6200, pager 600.

OTHER MEETINGS

August 17-19 in Washington, DC. The Centers for Disease Control, Food and Drug Administration, and Occupational Safety and Health Administration sponsor "Frontline Health-Care Workers: A National Conference of Device-Mediated Bloodborne Infections" at the Hyatt Regency Washington on Capitol Hill. Contact Laura Timperio, PACE Enterprises Inc, 17 Executive Park Dr NE, Suite 200. Atlanta GA 30329. (404) 633-8610, fax (404) 633-8745.

September 3-4 in Overland Park, Kansas. The Respiratory Therapy Department at the University of Kansas Medical Center presents its 24th Annual Respiratory Care Postgraduate Symposium, "Respiratory Care Crisis Intervention." AARC President Bob Demers presents the keynote address, "In Harm's Way." Contact Robin Dickerson RRT, Respiratory Therapy Services, University of Kansas Medical Center, 3901 Rainbow Blvd, Kansas City KS 66160-7282. (913) 588-3335.

September 9-11 in Captiva Island, Florida. The Suncoast Pulmonary Seminar is held at the South Seas Plantation. Presentations cover pediatric, neonatal, critical care, and management topics. Speakers include Neil MacIntyre MD, George Burton MD, Rich Branson RRT, and Judy Tietsort RN RRT. Contact Robert Sobkowiak (813) 627-2525.

September 18 in Beaumont, Texas. The Respiratory Care Department of St Elizabeth Hospital presents its 4th Annual Educational Seminar. National and local speakers lecture on a wide variety of topics for practitioners of all specialty areas. Contact Greg Rodgers RRT (409) 899-7065.

September 21-22 in Ann Arbor, Michigan. The University of Michigan Medical School's Department of Internal Medicine presents "Update on Pulmonary and Critical Care Medicine." The course features updates on controversies and evolving technologies in pulmonary and critical care medicine. Contact Edwina Borde, Conference Registrar, Towsley Center for Continuing Medical Education, Department of Post Graduate Medicine, University of Michigan Medical School, PO Box 1157, Ann Arbor MI 48106-1157.

October 9-10 in Vail, Colorado. The Colorado Advanced Life Support Committee holds its Biennial Resuscitation Conference, "Fresh Perspectives on ACLS and PALS," at the Westin Hotel. Join Joseph Ornato MD and James Seidel MD, among others, for state-of-the-art therapy and a review of the Dallas ECC Conference during the beautiful Fall season in the Rockies. Contact Colorado ALS, PO Box 440895, Aurora CO 80044. (303) 363-8380.

October 11-13 in Arlington, Virginia. The first National Asthma Conference is scheduled to hold a workshop specifically for respiratory care practitioners during the conference. The workshop has been approved for nine continuing education credits by the AARC, and is open to any respiratory care practitioner who wishes to attend. Contact NHLBI (301) 951-3275.

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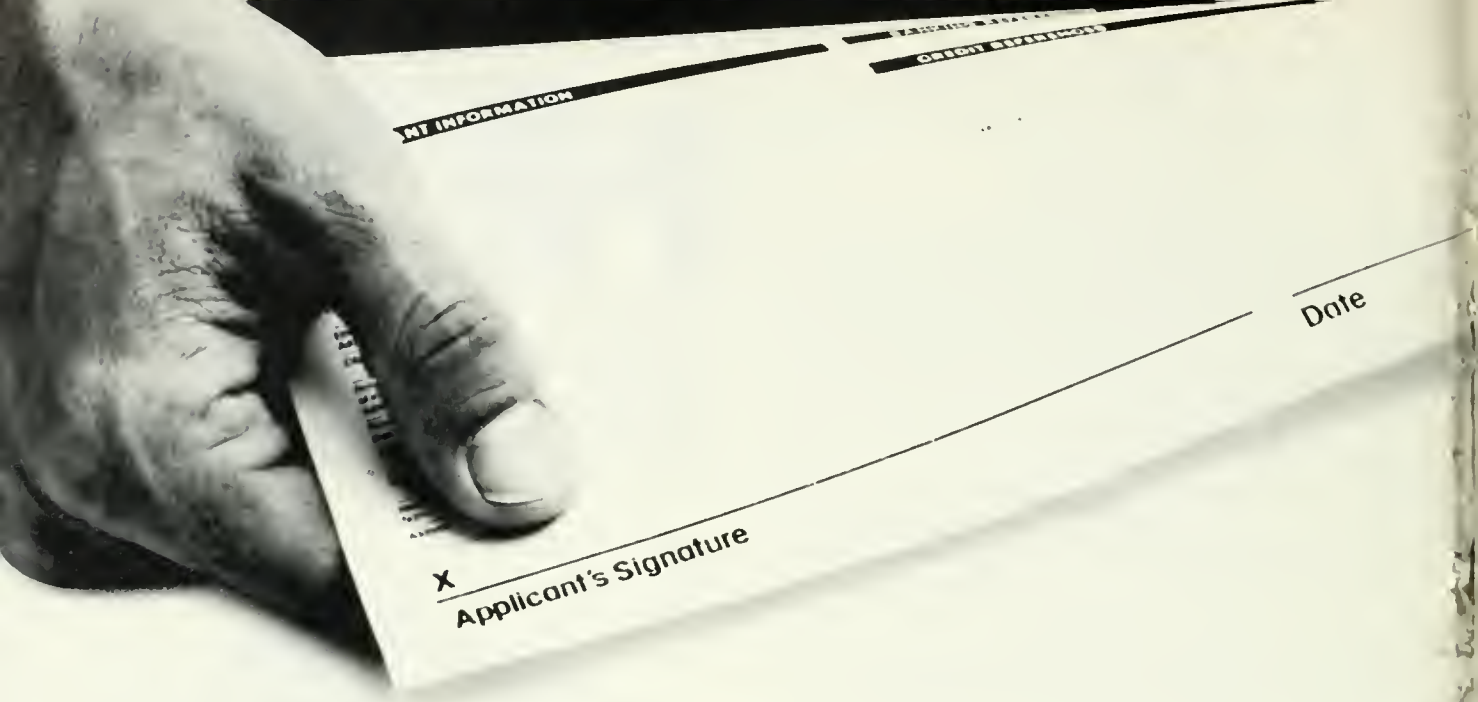

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7. ❑ Home Care Practice
8. ❑ School

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A. ❑ Respiratory Therapy
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C. ❑ Anesthesia Service
D. ❑ Emergency Dept

III. Specialty
1 ❑ Clinical Practice
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3. ❑ Critical Care
4 ❑ Clinical Research
5. ❑ Pulmonary Function Lab
6 ❑ Home Care/Rehab
7. ❑ Education
8. ❑ Management

IV. Position
A. ❑ Dept Head
B. ❑ Chief Therapist
C. ❑ Supervisor
D. ❑ Staff Technician
E. ❑ Staff Therapist
F. ❑ Educator
G. ❑ Medical Director
H. ❑ Anesthesiologist
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